

OBJECTIVE ASSESSMENT
OF
MATURATION OF POST-BURN HYPERTROPHIC SCAR
-- A LONGITUDINAL STUDY

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- A Longitudinal Study

Acknowledgement	v
Abstract	vii
Chapter One INTRODUCTION	1
Chapter Two LITERATURE REVIEW	4
1 STRUCTURE OF SKIN	4
1.1 epidermis	
1.1.1 stratum corneum	
1.1.2 stratum lucidum	
1.1.3 stratum granulosum	
1.1.4 stratum spinosum	
1.1.5 stratum germinativum	
1.2 dermis	
1.2.1 collagen	
1.2.2 elastin	
1.2.3 reticulin	
1.2.4 fibroblasts	
1.2.5 ground substance	
1.3 dermo-epidermal junction	
1.4 skin appendages	
1.4.1 hair	
1.4.2 nails	
1.4.3 glands	
1.5 cutaneous vascular system	
1.5.1 cutaneous blood flow and its significance	
1.5.2 cutaneous lymphatic flow	
2 FUNCTIONS OF SKIN	24
2.1 protection	
2.2 sensation	
2.3 thermal regulation	
2.4 absorption	
2.5 protection against ultraviolet radiation	
2.6 storage	
3 BIOMECHANICS OF SKIN	28
3.1 skin elasticity and the physical variation	
3.2 mechanical properties	
3.2.1 tensile strength	

3.2.2	distensibility	
3.2.3	Young's modulus	
3.2.4	visco-elastic character	
3.2.5	hysteresis	
3.3	fibre orientation	
3.4	mechanical considerations	
3.5	physiological factors	
3.6	clinical application	
4	PHYSIOLOGICAL RESPONSE OF HUMAN SKIN	47
4.1	response to mechanical loading	
4.1.1	triple response	
4.1.2	reactive hyperaemia	
4.2	thermal response	
4.2.1	skin temperature	
4.2.2	response to heat	
4.2.3	response to cold	
4.3	local tissue response to burn	
	Chapter Three BACKGROUND OF THE PRESENT STUDY	55
1	BURN INJURIES	55
1.1	nature	
1.2	depth	
1.3	extent	
1.4	location of burn	
1.5	age	
1.6	associated major trauma, inhalation injury	
1.7	general health status	
2	WOUND HEALING PROCESS	65
2.1	role of collagen in wound healing	
2.2	role of oxygen in wound healing	
2.3	role of fibroblasts and myofibroblasts in wound healing	
2.4	role of mast cells in wound healing	
3	HYPERTROPHIC SCAR	71
3.1	aetiological factors	
3.1.1	age	
3.1.2	time for wound healing	
3.1.3	racial factor	
3.1.4	depth of injury	
3.1.5	location	
3.1.6	tension	
3.2	characteristics	
3.3	pathogenesis of hypertrophic scar	
3.3.1	blood flow	
3.3.2	tissue gas	

3.3.3	filamentous material	
3.3.4	mast cells	
3.3.5	chondroitin sulfate	
3.3.6	enzyme proline hydroxylase	
3.4	histopathology	
3.5	response towards pressure	
4	TREATMENT OF POST-BURN HYPERTROPHIC SCAR AND THEIR RESPONSE	92
4.1	surgery	
4.2	radiotherapy	
4.3	ultrasonics	
4.4	chemotherapy/ intralesional injection of steroid	
4.5	pressure therapy	
4.6	topical silicone gel	
4.6.1	mechanics	
4.6.2	bacteriology	
4.6.3	water-vapour transmission rate	
4.6.4	appearance in the Scanning Electronic Microscope	
4.7	prevention of hypertrophic scar and scar contracture	
5	ASSESSMENT TOOLS FOR HYPERTROPHIC SCAR AND THE CLINICAL APPLICATION	105
5.1	clinical observation of the appearance	
5.2	ultrasonography and thickness	
5.2.1	ultrasound	
5.2.2	pulse-echo distance measurement	
5.2.3	echo generation	
5.2.4	transducer beam pattern	
5.2.5	ultrasound instrumentation	
5.2.6	application of ultrasound in the study of hypertrophic scar thickness	
5.3	elastometry (Cutometer) and elasticity	
5.4	application of elastometry	
	Chapter Four OBJECTIVES & METHODOLOGY OF THE STUDY	123
1	Objectives of the study	123
2	Study subjects	123
3	Methodology	125
4	Assessment of thickness of hypertrophic scar	128
5	Assessment of visco-elasticity of hypertrophic scar	130
6	Clinical rating scale	133
7	Study of normal skin as control	133
8	Reliability of the ultrasound and cutometer measurement	134

Chapter Five RESULTS 136

1	Inter- and intra- examiner variations of the ultrasound and cutometer measurement	136
2	Comparison with normal skin control	138
3	Results of ultrasonographic measurements of thickness of hypertrophic scar and its correlation with the clinical grading	139
4	Results of Cutometer reading (visco-elastic properties) and the correlation with clinical grading	142
5	Observation from raw data	152

Chapter Six DISCUSSION 154

1	Measuring thickness with ultrasonography and clinical grading	157
2	Elastic properties of hypertrophic scar and clinical grading	158
3	The predictive value of the ultrasonography and elastometry through monthly longitudinal measurement	161
4	Inter- and intra- examiner reliability of the ultrasonography and elastometry in the assessment of post-burn hypertrophic scar	164
5	The use of a composite "Visco-elasticity-Thickness Chart" and case studies	165
6	Limitations of the study	182

Chapter Seven CONCLUSION AND RECOMMENDATION

FOR FURTHER STUDY 184

REFERENCES 187

APPENDICES 197

Appendix 1	Patients' record	197
Appendix 2	Record of the scars	200
Appendix 3	Clinical Grading of the hypertrophic scar	202
Appendix 4	Measurement of the visco-elastic properties	204
Appendix 5	Ultrasonic measurements of the hypertrophic scars	235
Appendix 6	List of graphs	237
Appendix 7	List of figures	238
Appendix 8	List of tables	240

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Abstract

Hypertrophic scars are rigid and inextensible. After burn injury, many patients will develop hypertrophic scar. The severity is associated with the mode of injury, extensiveness and depth of the wound, and the time taken for the wound to heal. Hypertrophic scar is a sequelae of abnormal proliferation of collagen tissues resulted in an abnormal balance between the synthesis and degradation of collagen fibres.

During the maturation process, the thickened, inelastic hypertrophic scar will undergo certain change in the appearance. Clinical practice usually use only subjective description of the change in the appearance such as thickness, pliability and colour. An objective and valid assessment tool will be an essential adjunct to the clinical observation and would help in the documentation of the change of hypertrophic scar and in monitoring the effect of different treatment modalities.

This thesis described the application of elastometry and ultrasonography in the assessment of hypertrophic scar. A longitudinal study was conducted to measure the change of hypertrophic scar with the above-mentioned devices.

The Introduction provides a background to the understanding of the problem and emphasizing the essence of having an objective assessment tool. This chapter mentions the objectives of the study.

Chapter 2 provides the basic anatomy and physiology of skin which is a prerequisite to the understanding of the hypertrophic scar. Chapter 3 presents the background of the study. The content includes role of collagen, oxygen, mast cells, fibroblasts and myofibroblasts in the wound healing process. The formation and maturation of hypertrophic scar, the aetiology, characteristics, pathogenesis and histopathology of scar are also inclusive. This chapter also summarizes the treatment of hypertrophic scar. The application of the Elastometry and Ultrasonography in clinical practice are also described.

Objectives and methodology were described in Chapter 4. Sketching of the image were presented next to the ultrasonograph to aid the description of the measurement; graphic presentation of cutometer were reviewed in this chapter to explain the different values of the visco-elastic characters.

Chapter 5 provides the results of the study. The objective measurement of visco-elasticity (with elastometry) and thickness (with ultrasonography) was compared with the traditional clinical grading. Normal skin of patients was used as control for comparison. The inter- and intra- examiner reliabilities were studied with repeated measurement of hypertrophic scar.

The result was discussed in Chapter 6 with literature support. The high correlation of the measured thickness with the clinical grading supported that the use of ultrasonograph is reliable in the measurement of hypertrophic scar with a greater sensitivity.

For the use of the cutometer (the assessment of visco-elasticity of hypertrophic scar), value of R8 representing the visco-elastic properties is recommended to document the change and remodeling of hypertrophic scar. A cut-off point ($R8 = 0.193$) was also found to distinguish the pretty firm and active scar with significance.

R0 is also suggested to document the change of skin excursion in response to the negative pressure with longitudinal follow up.

A composite "Visco-elasticity-Thickness Chart" was suggested. The application is illustrated with 5 cases. The scar included chronic, active scar, mature scar and skin graft.

In Chapter 7, this thesis concluded with the recommendation of objective assessment with the ultrasonography and elastometry supplementary to clinical grading. Limitations of the study are also considered.

Chapter one: INTRODUCTION

After burn injury, the wound will eventually develop into hypertrophic scar. The dynamic balance of synthesis and degradation of collagen fibres is upset. The hypertrophic scar will undergo a process of proliferation, retraction and contraction, remodeling and getting mature. During this process, if the scar stretched across joints, it will develop into joint contractures and deformities. This could limit the active range of motion and hinder the limbs' functions. The period for hypertrophic scar becoming mature takes 18 months to several years.

Pressure therapy is the commonest conservative modality adopted in the treatment of hypertrophic scar. Various authors had studied the effect of pressure in the treatment of hypertrophic scar (Larson et al 1973, Ketchum & Cohen 1974, Kischer et al 1978, Cheng et al 1986, Linares 1996). Other treatments of post-burn hypertrophic scar include surgical excision with appropriate skin graft; radiotherapy, ultrasound, intralesional injection of steroid. Adjunctive therapy includes application of topical silicone gel (Quinn 1987, Ahn et al 1991, McNee 1992, Carney et al 1994). Anyhow, the principle is to hasten the maturation of hypertrophic scars.

Several studies had been conducted to prove the effectiveness of different modalities in the treatment of hypertrophic scar. However, the tools of assessment were varied. This leads to sophistication in prioritizing the effectiveness among the treatment modalities. Hence, an objective and globally accepted assessment tool is essential to monitor and document the change of maturation of hypertrophic scar. The

change of appearance is generally accepted and used in the clinical practice. It is non-invasive, fast to implement. The scorings still rely on the subjective perception of the clinicians. The inter-rater reliability is proved satisfactorily (Baryza & Baryza 1995). Yet, the validity is uncertain.

In this thesis, two instruments are introduced to document the morphological change of hypertrophic scar. The use of ultrasonography in the assessment of thickness of hypertrophic scar is analyzed. The study also includes the use of cutometer, an elastometry that provides a constant, non-invasive suction pressure to measure the visco-elasticity of hypertrophic scar.

As the hypertrophic scars become mature, the mechanical properties will improve gradually. 81 scars selected from 18 patients were longitudinally studied for 15 months respectively. The scars were assessed with clinical grading, ultrasonography and elastometry.

The result of the cutometer and ultrasound measurements were analyzed in comparison with the clinical grading. Finally a composite "Visco-elasticity-Thickness Chart" is suggested supplementary to clinical grading to aid judgment. For scar those were thick, persistent in thickness, with visco-elastic character R_8 greater than 0.193, close monitoring is essential and recommended.

The objectives of the study are as following:

1. To study the reliability of the ultrasonography and elastometry in the assessment of post-burn hypertrophic scar
2. To study the predictive value of the ultrasonography and elastometry through monthly longitudinal measurements.
3. To find out the correlation of the result with traditional clinical grading.

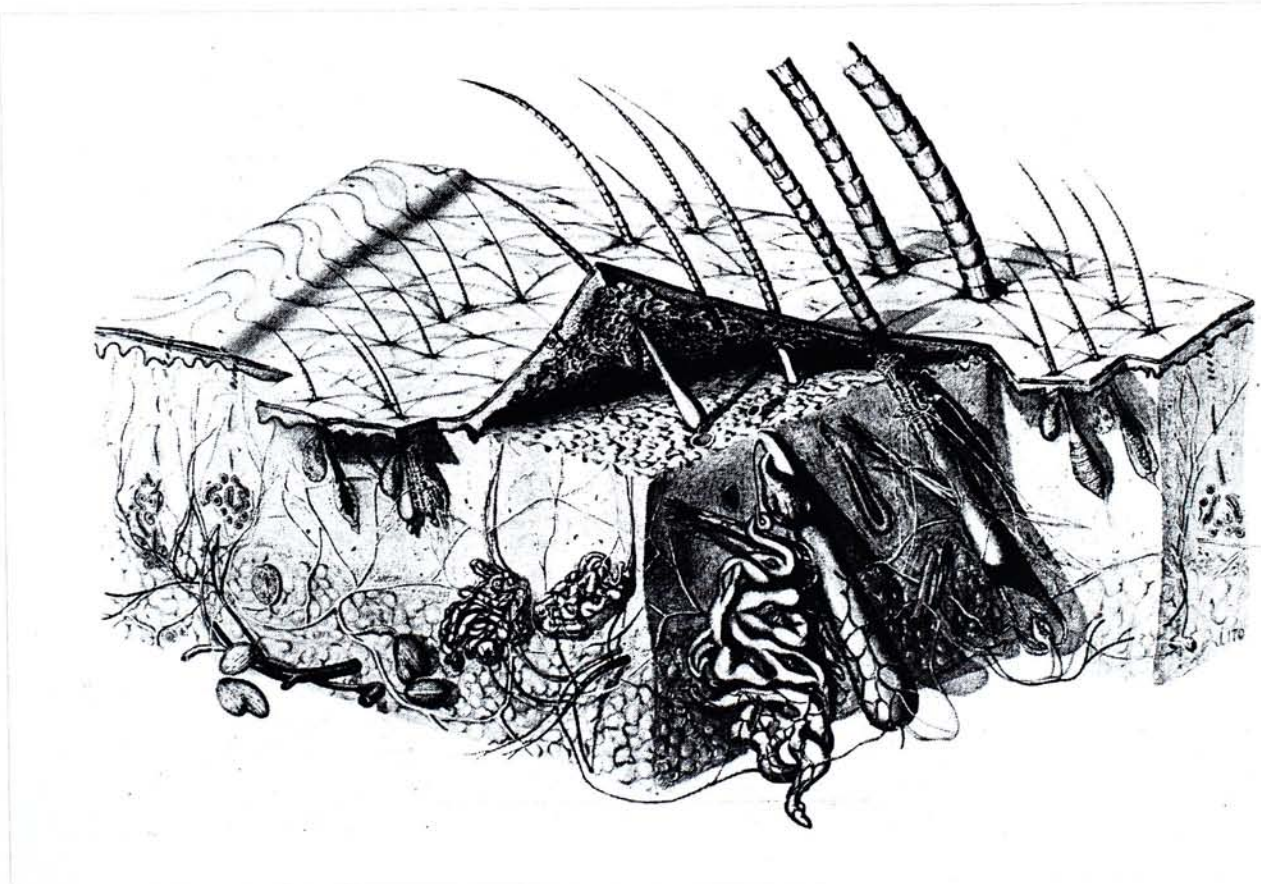
Chapter Two : LITERATURE REVIEW

A knowledge of skin physiology is fundamental to our understanding of post-burn hypertrophic scar. This chapter describes the basic anatomy and physiology of skin which formed an important basis for the understanding of hypertrophic scar.

1 STRUCTURE OF SKIN

The skin receives one-third of the blood circulating through the body. It is elastic, regenerates, and functions in sensation, protection, thermoregulation and secretion. (Fig. 1. Diagram of human skin, Montagna & Parakkal (1974) *The Structure and Function of Skin*, p.7)

Fig. 1



The skin consists of two layers, the epidermis and the dermis.

1.1 Epidermis

This is the outer layer of the skin composing five layers of stratified squamous epithelial cells (Jacob, Francone, & Lossow 1978).

From superficial to deep, they are :

1.1.1 Stratum corneum (horny layer)

This is the outermost layer of the epidermis and consists of dead cells completely filled with a protein called keratin. They are commonly called keratinized cells and are continuously shed, requiring replacement. The stratum corneum consists of 20 per cent water. It is composed of flattened cells resembling scales. It serves as a physical barrier to light and heat waves, microorganisms, and most chemicals.

1.1.2 Stratum lucidum (clear, hyalin layer)

Stratum lucidum, lying directly beneath the stratum corneum, is a layer of one to five cells thick, consisting of transparent, flattened, dead or dying cells, usually lacking nuclei.

1.1.3 Stratum granulosum (granular layer)

The stratum granulosum is thought to be active in keratinization, a differentiation process in which cells manufacture keratin and lose their nuclei, becoming more

compact and brittle. Uitto, Oikarinen & Thody (1986) defined keratinization should refer only to the synthesis, accumulation and aggregation of the keratin filaments.

The stratum granulosum composes of two to five layers of flattened cells, providing a transition into the subjacent layers. Granules accumulate in the cells, giving the layer its name; however, the granules do not contribute to skin color.

1.1.4 Stratum spinosum (prickly layer)

The Stratum spinosum consists of several rows of "prickly" cells, polyhedral in shape. The cells are spiny, hence the name, prickle cells. In some classifications this layer is included with the stratum germinativum as the malpighian layer.

1.1.5 Stratum germinativum/ stratum basale (basal, regenerative layer)

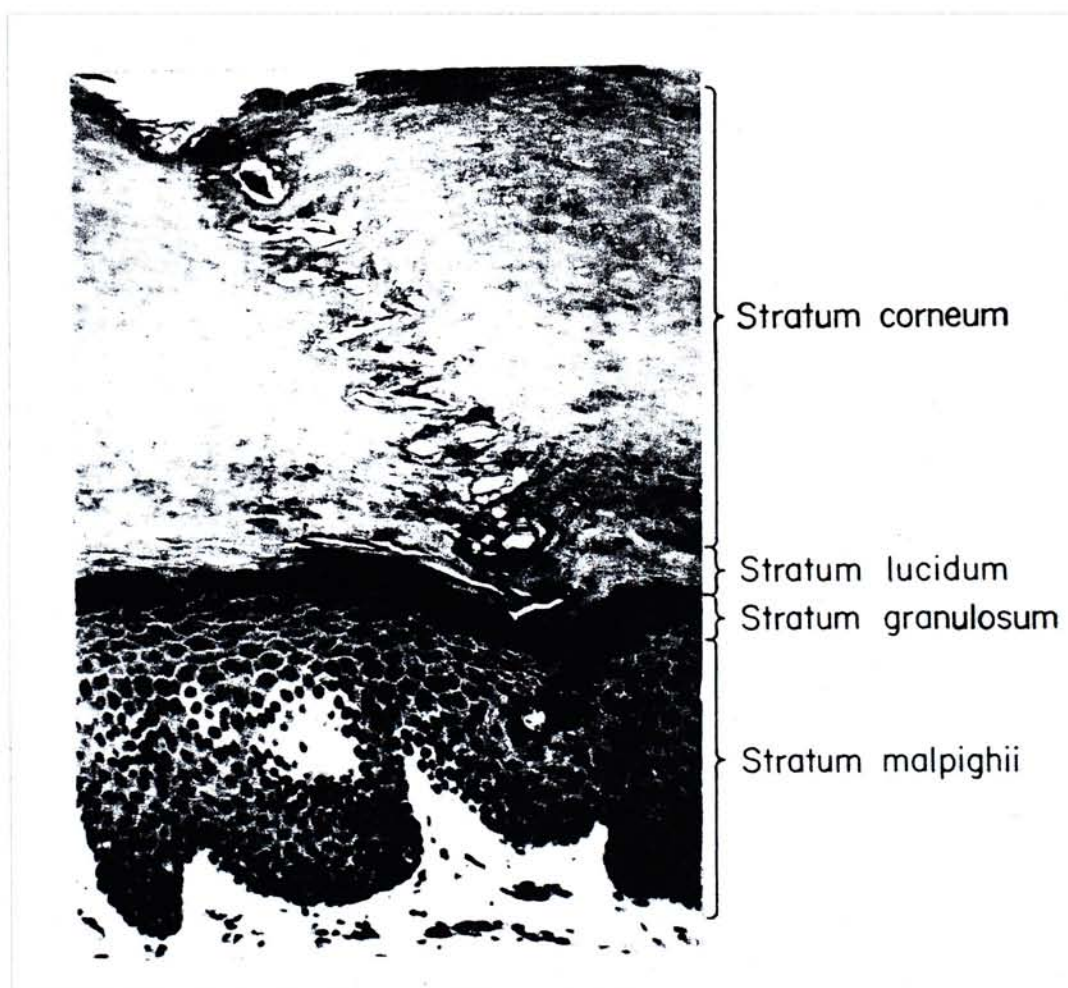
This is the deepest and most important layer of the skin contains cells capable of mitotic division. When new cells are formed, they undergo morphologic and nuclear changes as they move toward the most superficial layer. Simultaneously, these cells give rise to all outer layers of the epidermis. The epidermis will regenerate only so long as the germinativum remains intact. The basal layer of these generative cells rests on a basement membrane which offers further protection from the

environment. The stratum germinativum consists of 70 per cent water, as compared to 20 per cent water in the stratum corneum.

According to Montagna & Parakkal (1974), the stratum Malpighii is conventionally subdividing into the one-cell stratum germinativum and the stratum spinosum.

Fig. 2 Epidermis from the palm showing all of its layers .

Montagna & Parakkal (1974) The Structure and Function of Skin, p.20

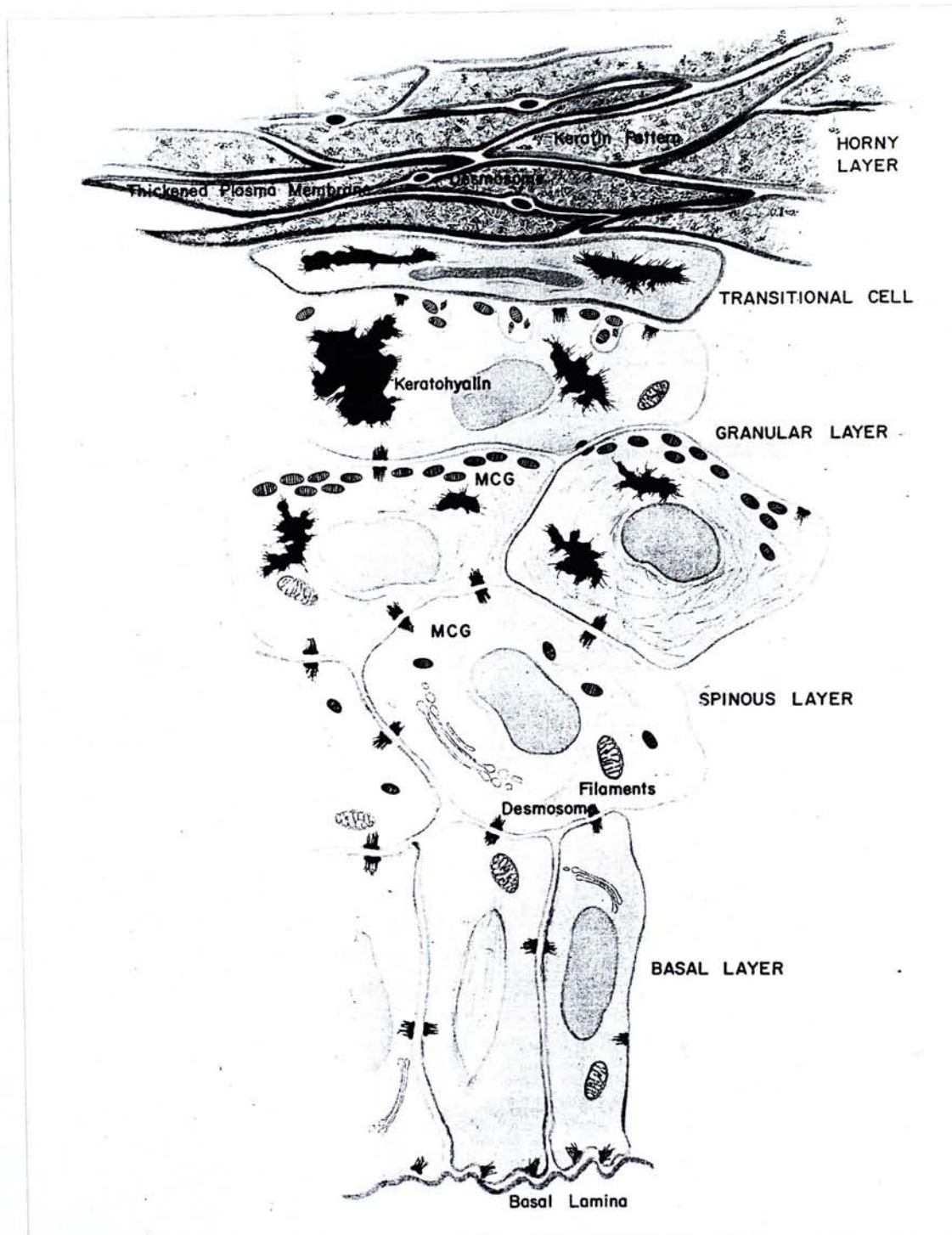


The epidermis composes mainly of keratinocytes. The basal layer of the epidermis has a permanent population of germinal cells whose progeny undergo specific patterns of differentiation. As epidermal cells migrate upward from the basal layer, they lose their mitotic potential to a great extent and begin to synthesize such specific constituents as fibrillar and amorphous proteins, keratohyalin, and membrane-coating granules. The surface becomes modified, and finally their nuclei and cytoplasmic organelles are resorped.

It is during this process of epidermal differentiation that the synthesis of keratin occurs, and it is these proteins which provide the structural and chemical integrity of the stratum corneum. With reference to Uitto, Oikarinen & Thody (1986), the epidermis can also respond locally to wear and wounding by increased keratinocyte proliferation producing more keratin.

After the cells have completed differentiation and become part of the protective system in the stratum corneum, on their way up to the surface, are eventually exfoliated. Thus, in the cycle of every epidermal cell, three distinct phases namely mitosis, differentiation, and exfoliation can be discerned (Montagna & Parakkal, 1974).

Fig. 3 Schematic diagram showing the layering of the epidermis. The basal cells are mitotically active. The differentiation products are shown in the different layers. The fully cornified cells are packed with a filament-matrix and show the "keratin pattern". Montagna & Parakkal (1974) *The Structure and Function of Skin*, p.46



1.2 Dermis

The dermis, lying directly beneath the epidermis, composed of connective tissue containing white collagenous and yellow elastic fibers. Blood vessels, lymph vessels, nerves, glands, and hair follicles are embedded in the dermis. The dermis is divided into a superficial papillary layer and a deep reticular layer. The papillary layer contains many conical eminence, the papillae, which project perpendicularly into corresponding depressions in the epidermis. Within each papilla is a capillary loop, furnishing a blood supply for the epidermis. The reticular layer is composed for the most part of white fibrous tissue which support the blood vessels and other structures contained in it. The reticular portion of the dermis rests on the subcutaneous connective tissue (Jung & Earle 1955).

Montagna & Parkkal (1974) provided some basic facts about the dynamics of connective tissue and their relation to dermal function. The dermal matrix contains few cells (more in the upper papillary layer than in the lower reticular), which are predominantly fibroblasts with the potential to produce most the components of he extracellular matrix. More abundant are the mast cells; in addition, histocytes or macrophages, melanocytes, and extravasated leukocytes are often found. During dermal remodeling, macrophages participate in collagen degradation. In Man, the whole mass of dermis constitutes from 15 to 20% of total body weight. The versatility of the dermis is seen in its range of functions, from ion exchange to protection from mechanical injury. It provides nourishment to the epidermis and interacts with

it during embryogenesis, morphogenesis, repair and remodeling. Its various properties stem primarily for the matrix of extracellular connective tissue, the ground substance, and the fibrous proteins.

Histological examination of different levels of dermis reveals that the organization of the connective tissue fibers is different in papillary dermis as compared to that in mid or lower reticular dermis. The papillary dermis contains fibers of smaller diameter and show more loose packing arrangement than the fibers in the reticular dermis. This features may reflect differences in the biochemical composition of the reticular and papillary dermis. Differences in the interactions of the components and their relative quantities can explain some of the variation noted in the physiological properties of the skin at different locations.

1.2.1 Collagen

Collagen is the principal fibrillar component of the dermal connective tissue, constituting approximately 70-80% of the dry weight of the dermis (Uitto et al 1986). This structural body protein provide the strength and stiffness to dermal tissue,. Collagen fibers, with diameters from 2 to 15 μm , form finely woven networks in the papillary layer of he dermis or thick bundles paralleling the skin surface. These bundles are relatively inextensible and non-elastic, and thus give dermis a high tensile strength.

Five major types of collagen namely type I to type V could be identified. They vary only in the amino acid constructs. Type I collagen is found in adult dermis, fascia, and bone; type II collagen in adult cartilage; type III collagen in embryonic connective tissue, aorta, deep dermis, and wounds; and types IV and V collagen in basement membrane.

Only types I and II collagen are found in dermis and are involved in wound healing. The relative proportions of types I and III collagen appear to vary with chronological age, such that type III collagen is gradually replaced by type I collagen with aging. The diameters of collagen fibers appear to systematically vary throughout dermal depth. Type I collagen fibrils range from 100 to 500 nm and type III collagen fibrils from 40 to 60 nm in diameter, and the proportion of larger size fibrils ultimately determines the skin tissue tensile strength. Type III collagen is found at deepest levels.

From biochemical point of view, collagen can be described in a generic fashion as being composed of three polypeptide alpha chains arranged in a triple helix, several of which are cross-linked together to form collagen fibrils. While both type I and type III collagen have the same procollagen precursor, differentiation occurs such that type I collagen is formed by two identical alpha-1 chains and a third alpha-2 chain, and type III collagen consists of three identical alpha-1 chains. The

collagen fibrils then twine together to form collagen fibers. Dermal collagen fibers, inherently inelastic and inextensible, form a loose interlacing and deformable network, able to align in directions that accommodate applied stress, and therefore allow skin to stretch (Price 1990).

In adult skin, type I collagen comprises about 80% of the total collagen and about 15% is type III. During wound healing or skin development in the fetus, the deposition of type III collagen precedes the synthesis of type I collagen (Uitto et al 1986).

1.2.2 Elastin

Elastin is a highly hydrophobic structural protein providing elasticity in the dermis and comprising only 2 % of the protein in dermis. Elastin, lipids, and glycoproteins bind to form microfibrils that serve as the scaffolding for future fiber orientation. Elastin is thus able to inherently and functionally be elastic, providing good recoil, and extensible, providing tissue length by unwinding the fiber wave. (Price 1990)

Under light microscope, elastic tissues of normal human skin can be divided into three different types of fibbers - oxytalan, elauninm and the elastic fibbers. The oxytalan fibbers are the most superficial, thin and directly perpendicular to the dermal-epidermal junction. These fibers are formed of bundles of tubular microfibrils, 10-12 nm in diameter.

The oxytalan fibers are connected to a plexus of fibers. The elaunin fibers in turn are connected with thick elastic fibers of the lower papillary and the reticular dermis.

The elastic fibres consist of two distinct protein components. By electron microscopy the major component has an amorphous appearance, and this part of the elastic fibre represents elastin, a well-characterised connective tissue protein. This amorphous elastin core is surrounded by distinct fibrillar structures which have a regular diameter of 10-12 nm; these fibrils are called the elastic tissue microfibrillar component. The relative proportions of these two components change during development. During early embryonic development, most of the elastic fibres consist of the microfibrils which are replaced by elastin at the later stages of development. In fact, in a mature, fully developed elastic tissue, well over 90% of the total content consists of elastin (Uitto et al 1986).

1.2.3 Reticulin

Reticulin is the least prevalent fiber found in the dermis. It appears as fine-branching fibers, composed of sparsely distributed fibrils, with the typical 640 Å axial periodicity of collagen, 0.2 to 1 µm in diameter. Reticulin also contains large quantities (10.9%) of firmly bound fatty acid and can thus be considered a lipoglycoprotein. Reticulin fibers form a supporting framework for many organs and glands. Reticulin

may also form an early framework upon which collagen fibers are laid down and hence, aggregated. They have been found in very small numbers in normal skin and in significant numbers in early phases of the healing wound. (Price 1990; Montagna & Parkkal 1974).

1.2.4 Fibroblasts

Under normal conditions, fibroblasts are the most numerous cells in connective tissue, responsible for the formation of collagenous and elastic fibers and the amorphous ground substance (Montagna & Parkkal 1974).

In normal skin, connective tissue turnover is a continual process. All components of connective tissue are phagocytized and replaced with new cells perpetually. The fibroblast is responsible for production of replacement collagen throughout life. With wounding, the process of biosynthesis is potentiated. In the inflammatory phase, macrophages digest denatured collagen, thus removing non-collagenous proteins, and excrete useful amino acids and simple sugars for future synthesis. During the fibroblastic phase, about day 10 after wounding, fibroblasts, which have increased in population, begin synthesizing at a higher rate (Price 1990).

1.2.5 Ground substance

This is the structureless portion of connective tissue lies outside the cells, fibers, blood vessels, and nerves; all other dermal components are embedded in this amorphous matrix. Functionally, the ground substance provides density to tissue and reduces friction between connective tissue fibers during tissue stress or strain.

The ground substances appears as to be a multi-component system of *substances derived from the blood* (such as water, inorganic ions, blood sugars, blood proteins, and urea); *metabolic products of parenchyma cells*; and *metabolic products of connective tissue cells* (the soluble precursors of the fibrous proteins, and the proteoglycans, glycoproteins, and complexes formed from these).

The substrates of ground substance are glycoproteins and mucopolysaccharides, particularly glycosaminoglycans (GAG). Most likely, saccharide chains in the chondroitin sulphate - protein complex cross-link to collagen fibers, and the cross-linkage allows for departmental or small tissue area shifts in pressure, much like an hydraulic dampening, therefore contributing to integrity and force transduction in skin (Price H. 1990).

The ground substances serve as the internal milieu, the immediate environment of dermal and epidermal cells, and an extension of the

vascular system. For the metabolites of all cells, mesenchymal or epithelial, which are separated from their blood supply by connective tissue, must pass through this bound water (Montagna & Parkkal 1974).

In accordance with Uitto et al (1986), the physiological importance of dermis is to provide tensile strength to the skin. This property of dermis is primarily attribute to the fibrillar components of the connective tissue, collagen and elastin, which exist in dermis in a fibrous interwoven meshwork.

1.3 Dermo-epidermal junction

The basement membrane zone, which lies between the epidermis and dermis, is a complex structure comprised of several different macromolecular components. Morphologically, the basement membrane at the dermal-epidermal junction can be divided into two parts. The upper half is known as lamina lucida, and the lower counterpart is known as lamina densa or basal lamina. The plasma membrane of the basal cells has specialized attachment structures - hemidesmosomes. Fine anchoring filaments traverse the lamina lucida and are embedded in the lamina densa. Below the lamina, the sub-basal region contains anchoring fibrils, dermal microfibrils and single small collagen fibers which are often enmeshed with loops of anchoring fibrils. The anchoring fibrils extend from the dermis to be embedded in the lower part of the lamina

densa, thus playing an important role in the attachment of the dermis to the basement membrane zone (Uitto et al 1986).

The dermal-epidermal junction postulated at least three functions :

- the basement membrane zone serves as an attachment and adherence site between the dermis and epidermis;
- the basement membrane zone appears to provide mechanical support for the epidermal cells;
- the basement membrane zone serves as a semi-permeable barrier or filter which regulates the transfer of materials and cells across the dermal epidermal junction.

1.4 Skin appendages

The appendages associated with the skin include hair, nails, sebaceous glands, and sweat glands.

1.4.1 Hair

Hair covers the entire body except the palms, soles, and portions of the genitalia; each unit of hair is composed of three parts - the cuticle, cortex, and medulla naming respectively from the outermost portion, the principal portion and the central axis of the hair. The visible portion of the hair is the shaft. The cells at the base of the root, which develops into the shaft. Hair grows in a tubular invagination of the epidermis called the hair follicle, which is surrounded by dermal connective tissue.

When the arrector pili muscles (bundles of smooth muscle fibers attached to the hair follicles) contract, the skin assumes a so-called "goose flesh" appearance where hair is sparse and results in a certain degree of "hair standing on its ends" where the hair is prominent.

1.4.2 Nails

The nails, a modification of the horny epidermal cells, are composed of hard keratin. Air mixed in the keratin matrix forms the white crescent, the lunula, at the proximal end of each nail. The nail plate arising from the proximal nail fold and attached to the nail bed grows approximately 1mm. per week unless inhibited by disease.

1.4.3 Glands

Sebaceous glands

Sebaceous glands generally arise from the walls of hair follicles and produce sebum, the oily substance primarily responsible for lubrication of the surface of the skin. When the cell disintegrates, sebum is secreted along the hair shaft onto the surface of the skin, providing a cosmetic gloss.

Sebaceous secretion is under the control of the endocrine system, increasing at puberty and in late pregnancy and decreasing with advancing age. The pubertal increase contributes to the problem of

acne in adolescents, and diminution is responsible for the relative dryness of the skin in later life.

Sweat glands

Sweat glands are simple tubular glands found in most parts of the skin, and most are not associated with hair follicles. Each consists of a secretory portion and an excretory duct. The secretory portion, located in the dermis tissue, is a blind tube twisted and coiled on itself. From the coiled secretory portion, the excretory duct spirals toward the surface. Each glandular tube is lined with secretory epithelium continuous with the epidermis. The secretory epithelium consists of two types of cells : *spindle-shaped, contractile, myoepithelial cells* attached to the basement membrane, and *pyramidal cells* that secrete sweat, resting on top of the myoepithelial cells.

Ceruminous glands secrete wax, being found in the external meatus of the ear; while ciliary glands located in the eyelids, secrete ocular fluid. Both are considered to be modified sweat glands.

Sweating leads to loss of heat in the body owing to the fact that heat is required to evaporate the water in the sweat; thus, sweating helps to lower the body temperature. Sweating is initiated by the effect of elevated blood temperature on cerebral centers (Jung & Earle 1945, Jacob et al 1978).

1.5 Cutaneous vascular system

The vasculature needed by the epidermis include blood supply and lymphatic system.

1.5.1 Cutaneous blood flow and its significance

The capillary bed is the source of nutrition of the epidermis; contributes to skin color and thus to the function of display; moisturization and provides the cells that recognize foreign material invading the epidermis. It is part of the body's system for thermoregulation, and contributes to the vascular capacity of the entire organism, the peripheral resistance and control of blood pressure (Ryan 1993).

The normal blood supply to the skin consists of capillaries, mostly sited within the papillae of the upper dermis and therefore perpendicular to the surface. They have the shape of a hairpin loop and tend to present only their peaks to view. They are supplied by a deeper arterial system, and the overall pattern resembles a candelabra (fig. to show the shape of a candelabra) that drains into a rich subpapillary venous plexus. It is the subpapillary venous plexus that provides most of the color seen at the surface of the skin. Because the papillary vessels are the nutritional capillaries, they provide the tissue fluid that lies in the upper dermis and acts as an obscuring veil overlying the subpapillary venous plexus. The number of papillary vessels at the surface is approximately 60-70 per mm^2 .

When there is an increased demand by the epidermis, as occurs in wound healing or in disease like psoriasis, the papillary vessels tend to elongate and become more tortuous and have been likened to the tortuosity of cotton in cotton wool balls. A feature of such vessels is that they are closely applied to the epidermis, projecting into the papilla and almost completely surrounded by epidermis.

1.5.2 Cutaneous lymphatic flow

Cutaneous lymphatics are probably as extensive as blood vessels, but they have not been shown satisfactorily. After injection of radio-opaque substances, radiological techniques reveal a series of capillaries dead-ending in the papillary dermis and draining into a "subpapillary lymphatic plexus" that finally empties into deeper plexuses with valves. Lymph vessels progress centripetally via progressively larger ones, and filtering through lymph nodes, empty into the thoracic duct. Montagna & Parakkal (1974) suggested rather that the blind-ending lymphatic capillaries empty into a vast, unstructured network throughout the dermis. Larger vessels in the deeper portions of the dermis and in the hypodermis can be recognized by their numerous valves.

The lymphatic system is indispensable to blood circulation, which is at a relatively high pressure. Fluid passes out of arterial capillaries into the surrounding tissue because the pressure within the vessels is greater

than that in the tissue. Lymph fluids pass back into venous capillaries where pressure is low. Furthermore, because the osmotic pressure of plasma proteins is greater in the arterial vessels, they leak out into the tissues and cannot pass back into the circulation except through the sequent rise in osmotic pressure disturbs the balance of capillary filtration and causes the fluid to remain in the tissues; the result is edema. The principal role of the lymphatic system, then is to remove plasma proteins from extracellular spaces. Secondly, it removes particulate and antigenic materials from tissues.

The flow of lymph is slow and varies according to muscular activity. The one-way flow from the tissues into the lymphatic vessels is maintained by the direction of the valves.

2 FUNCTIONS OF SKIN

The skin functions in protection, sensation, thermal regulation, protection against ultraviolet radiation, storage and absorption.

2.1 Protection (protection against bacterial invasion and mechanical injuries)

The skin forms an elastic, resistant covering that protects man from his complex environment. It prevents the passage of harmful physical and chemical agents and inhibits excessive loss of water and electrolytes. Sebum secreted by sebaceous glands has antifungal and antibacterial properties and helps maintain the texture of the skin (Jacob et al 1978).

The skin plays a vital role in defense against invasion by microbes, presenting a physical and chemical barrier that is normally virtually impenetrable. If any micro-organism penetrate the outer defenses, the Langerhans' cells, deployed to detect such infiltration and to alert the inner defenses of acquired immunity. The second line of defense is the dermal inflammatory response which may use weaponry phagocytes and their products, and also the specific surveillance and recognition of acquired immunity represented by lymphocytes and their products (Friedmann 1986).

2.2 Sensation

There are specific receptors located in the skin sensitive to four basic sensations of pain, touch, temperature and pressure. Upon stimulation of a receptor, a nerve impulse is sent to the cerebral cortex of the brain, where the impulse is interpreted. The brain must interpret among degrees of stimulation and among combinations of stimulation, the latter of which result in sensations such as burning, tickling, and itching.

2.3 Thermal regulation

Heat is lost from the body by conduction, convection, radiation, and evaporation. These processes are regulated by nervous and chemical activation of the sweat glands, dilation and constriction of the cutaneous vessels. As the body needs to dissipate heat, blood vessels of the skin dilate, allowing more blood to come to the surface, with a resulting heat loss (Jung & Earle 1945, Jacob et al 1978).

The structural and physiological systems can modify and exchange energy with the surrounding environment. McEwan (1986) identified four ways :

- the cutaneous vascular supply
- the sweat gland
- the hair and hair coat
- the pigmentary processes in the hair and epidermis

Peripheral insulation is aided by the deposition of fat in dermal and subcutaneous stores.

2.4 Absorption

When the body exposed to the sun light, it will absorb the UVB portion (290-320nm) and convert the 7-dehydrocholesterol (a provitamin D₃) into vitamin D₃ through a process of photolysis. This non-enzymatic process take place primarily in the lower layers of the epidermis, the spinous layer and the basal layer (Hsia 1986).

2.5 Protection against ultraviolet radiation

Ultraviolet radiation initiates a multitude of photochemical reactions in biological matter. Most of these reactions induce detrimental effects in their target structure. In the skin, apart from the beneficial effect of photoconversion of

7-dehydrocholesterol into vitamin D₃, almost all known effects of ultraviolet are harmful and may have serious consequences such as cellular injury, changes of cellular kinetics, cell death and carcinogenesis.

Two natural defense mechanisms have evolved from human skin maintain the integrity against the constant impact of solar radiation : a protective and a reparative system. The protective system includes a number of structural components capable of scattering (reflecting) and of absorbing ultraviolet radiation. The most important of these are melanin, proteins in the viable epidermis and keratin in the stratum corneum, urocanic acid and some other aromatic chromophores. The repair system consists of at least three different types of mechanisms which are designed for the repair of cellular DNA :

excision repair, postreplication repair and photoreactivation (Hönigsmann & Thody 1986).

2.6 Storage

The stratum corneum and the dermis seems to have a storage property. Some topically applied substances appear to be stored in the skin and are released more or less rapidly after the source has been removed.

The reservoir can be loaded by application of substances under conditions that enhance percutaneous absorption - such as hydration under occlusion. Drying the skin slows down diffusion. The substance may be released slowly over a long period. Rehydrating the skin by occlusion can allow increased release of the substance (Hönigsmann & Thody 1986).

3 Bio-mechanics of skin

3.1 Skin elasticity and the physical variation

The mechanical characteristics of skin varies with body parts. Wright (1971) measured the skin-fold compressibility at the arm, calf, scapula, and waist. The result showed that while there was a decrease with age this decrement was not uniform over the body surface. In addition, changes in stiffness of the collagen and the architecture of the wicker-work change with age.

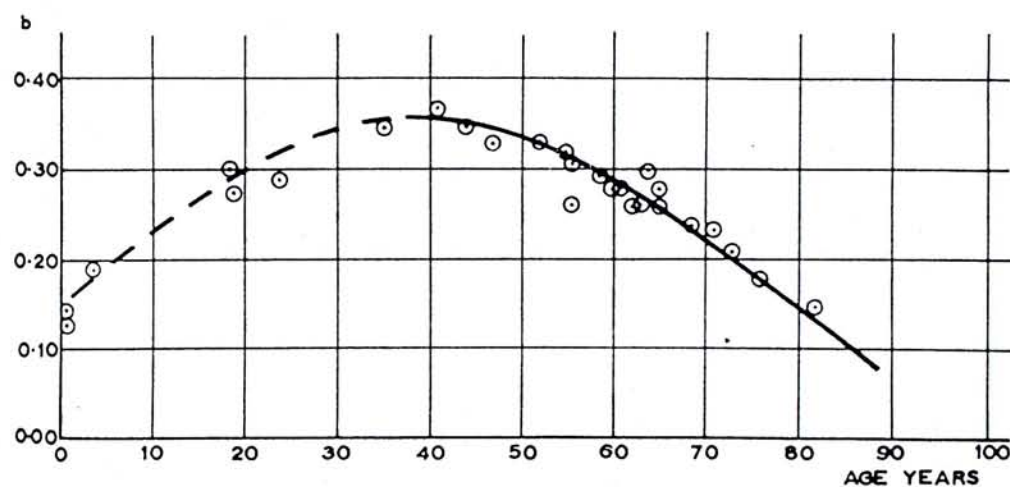
Sex

The tensile strength of female skin is less than that of male skin. Stress/strain measurements showed female skin to be more extensible, and the modulus of elasticity is significantly higher. Evidence showed that the relaxation of the tension in situ was greater in men than women, with value found 14% and 10.7% respectively.

Age

A decrease of the compressibility of skin plus subcutaneous tissue was found with advanced age. (Fig. 4 shows variation of elastic stiffness b with age of skin isolated from male abdomen, from Wright 1971, Biophysical Properties of the Skin, p. 445). With advancing age the number of cross-links in the collagen fibers are increased. There is a progressive rise in the modulus of elasticity of skin with age in both sexes. On the contrary, elderly subjects show the condition known as "transparent skin", there is a significantly lower modulus of elasticity.

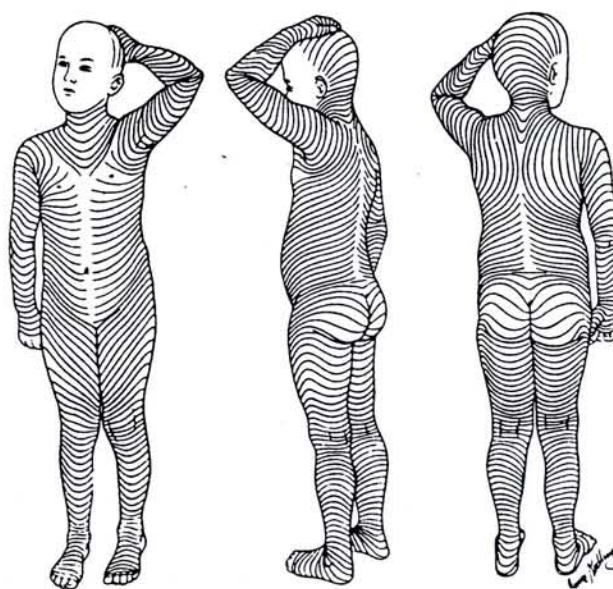
Fig. 4 Variation of elastic stiffness b with age of skin isolated from male abdomen,
from Wright 1971, *Biophysical Properties of the Skin*, p. 445



Site (natural lines of skin tension)

If the skin of a cadaver is punctured with a round awl, the holes are elliptical, the ellipses being oriented perpendicular to the lines of minimal extensibility of skin. These are known as Langer's lines. (Fig. 5 to show Langer's lines of the body)

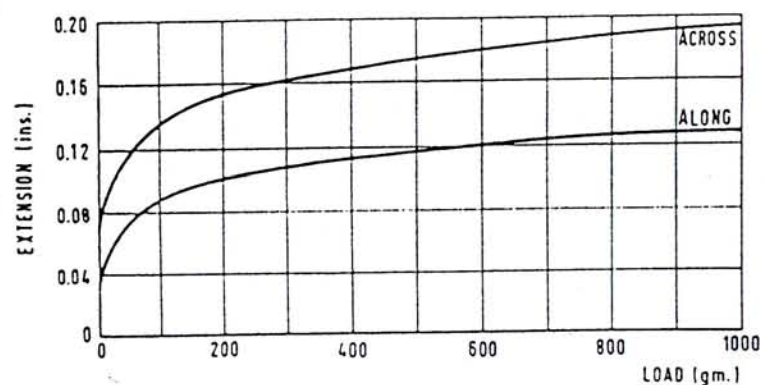
Fig. 5
Langer's lines
of the body



They represent directions of principle tension. Differences were found in the stress-strain curve of specimens taken along and across Langer's lines. A smaller extension of skin specimen taken in the direction of Langer's lines in comparison with across the Langer's lines can be obtained. It is because, across Langer's lines, a smaller number of fibers are extending, which have a greater orientated length than those in the direction along the lines. It is the architecture of the wicker-work which is changed. (Fig. 6 shows the stress/strain curves for abdominal skin from a woman age 88 taken along and across Langer's lines, Wright 1971)

Fig. 6

Stress/strain curves for abdominal skin from a woman age 88 taken along and across Langer's lines, Wright 1971, Biophysical Properties of the Skin p. 446



Regional differences in mechanical characteristics adapt skin to local demands and may reflect, the local structure of the dermal collagen and elastin networks. The orientation of fibers in the dermis also varies from one area to the next. Langer's lines follow the direction of preferential orientation of the fibers histologically. Along Langer's lines, collagen fibers do not have the typical meshwork arrangement but are oriented parallel to the lines (Montagna & Parakkal 1974).

3.2 Mechanical properties

Wright (1971) described skin as a nonlinear, nonhomogenic, viscoelastic material.

3.2.1 Tensile strength

The average tensile strength of human skin is 1.8 kg mm^{-2} . However, it varies with age :

$0.25\text{-}0.3 \text{ kg mm}^{-2}$ for infants up to 3 months

$0.53\text{-}1.4 \text{ kg mm}^{-2}$ for children 3 months to 3 years old

1.61 kg mm^{-2} for adults from 15 to 50 years of age

2.05 kg mm^{-2} for people from 50-80 years old

The tensile strength also depends on the rate of loading, (rapid loading giving a value many times that of static loading); sexes (pregnant women will have a different value of tensile strength), direction, and sites as different percentage of collagen will affect the properties.

3.2.2 Distensibility

The distensibility of skin decreases with increasing age. Skin taken from infants at birth shows a value of extension of 50-59%, 37-52% for children, and 24-48% for adults.

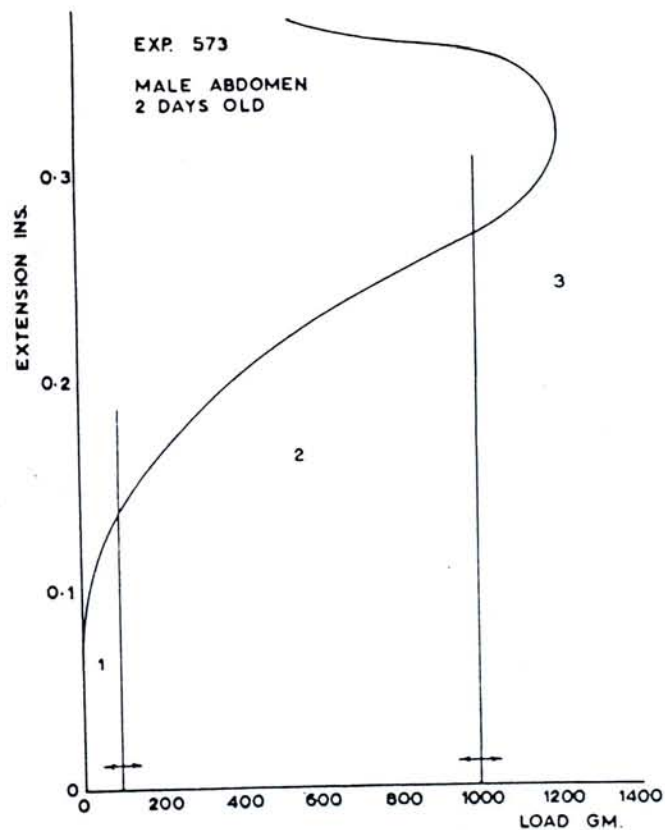
3.2.3 Young's Modulus

The stress/strain curve is not linear for skin, therefore, it is difficult to quote a Young's modulus for skin, though some have claimed that after 5% strain is achieved Hooke's law applies. The general form of the curve for various biological tissues is well established, commencing tangential to the extension axis, gradually bending toward the load axis. With a curve of this type it is imperative that the load at which the modulus is calculated.

A characteristic load-extension curve of skin is shown in Fig. 7.

Fig. 7

The load-extension curve of skin from male abdomen, Wright 1971, Biophysical Properties of Skin p. 441.



The extension process can be divided into three phases by boundaries at approximately 100g and 1000g. It has been shown histologically at :

First phase corresponds to the straightening out and orientation of the collagen fibers of the specimen and this process may be defined by the equation :

$$E = xy \log L$$

where E = extension, L = load and xy = constants.

Second phase of skin extension attributable to the extension of orientated collagen fibers, and characterized by the equation :

$$E = c + kL^b$$

where c , k , b = constants.

b reflected a specific property of the collagen fibers. This was shown theoretically and experimentally to be independent of the size of the specimen or the direction from which it was cut.

The constant k was shown to represent the conditions of the fiber meshwork and was governed by the length and area of the fibers.

Third phase of extension concerns yielding, when the skin gradually begins to extend more readily as individual fibers break down.

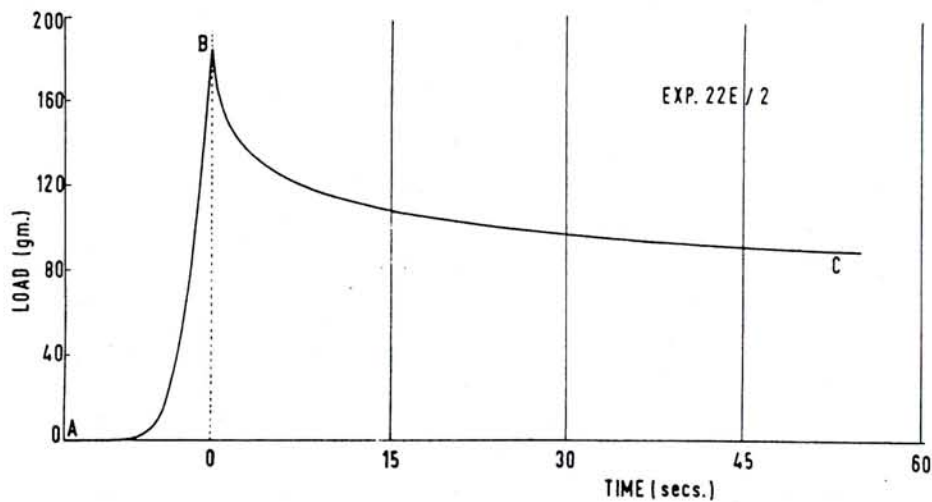
It was reported that when initial strains up to 20% of the unloaded length produced no significant increase in load, but that strains beyond about 40% required exceptionally high loads to produce further deformation. As an indication of the nonlinearity of skin, and the value of Poisson's ratio was generally in the region of unity, and hence viscoelastic features of skin is implicated.

The alignment of collagen fibers in the skin under stress was studied histologically. When pieces of postmortem abdominal human skin were stretched, the collagen fibers of the dermis became orientated in the plane of stress, and eventually showed fracture lines and then complete disruption. Usually the fibers is oriented, at low-load levels relaxation allowed the fibers to reassemble themselves in their normal random fession. At slightly greater loads, the fibers assumed a wavy pattern. Greater stress produced changes of orientation that were not reversed by relaxing the tissue.

3.2.4 Visco-elastic character

The skin is a viscoelastic material and is shown in Fig. 8. The viscoelastic character is shown by its marked stress relaxation when subjected to a constant load for a period.

Fig. 8 Stress relaxation of skin held at constant length (B to C)
after stretching to peak force (A to B),
Wright 1971, *Biophysical Properties of the Skin*, p. 443

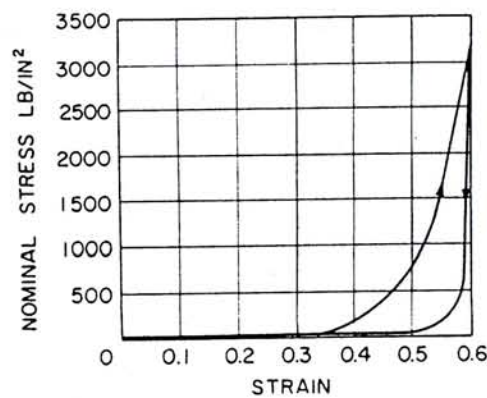


Stark (1977) studied the directional variations in the extensibility of human skin. The extensibility of human skin enables the body to move. However, the extensibility is very variable. It varies from one site to another on the same body; at any one site on one body, it may be quite different from that on the same site on another body; at any one site extensibility shows directional variations; finally, at any site and in any direction it decreases with age. The basic "stress-strain" curve can be obtained with skin and most soft tissue. The initial extension requires relatively little load but is followed by a terminal phase of high stiffness during which the skin requires very much higher increments in load to achieve similar increments in extension.

3.2.5 Hysteresis

Upon release of a stretched tissue in the physiologic range of elongation, the tissue will tend to return to its original length. However, this "unloading" curve is different from the loading curve and the curves are described as being noncoincident (as shown in fig. 9).

Fig. 9 Stress-strain curve, Montagna & Parakkal 1974,
The Dermis in The Structure and Function of Skin, p.131



When exposed to tension, skin "gives" until the slack is taken up. This slack results from the loose random arrangement of the collagen network at rest. When a load is applied, the fibers within the network undergo parallel alignment in the direction of the pull. Once this slack is taken up, further extension of the skin requires much higher tension. Great force is required to tear skin. Skin that is kept taut for long periods gradually undergoes "slippage", as if fatigued, and stretching

results. Hysteresis is defined as the delay in the retraction of the tissue to a shorter length, and characterized by the elastic recovery after termination of load (Montagna & Parakkal 1974, Reginald & Marllys 1994, Ennen et al) .

Collagen, elastin, nerve fibers, small blood vessels and lymphatics are the five basic components of skin. They intertwined with each other, covered by a layer of partially keratinized epithelium and transfixed at intervals by hair and the ducts of sweat glands. The networks are surrounded by interstitial fluid containing a varying amount of mucopolysaccharide ground substance which provide a semi-fluid environment. All the networks are mobile in their environment to varying degrees. In the palm and sole, the keratinous outer layer is relatively very thick, and probably plays a major role in defining the mechanical properties of the skin. Both the time-dependent mechanical effects and biological effects should be considered in studying the properties of skin (Kenedi 1980).

Skin can be classified as tissue with complex structure and interactive component behavior, in which elastin, collagen and ground substance interact to varying degrees at virtually every stage of loading.

Montagna & Parakkal (1974) quoted Tregear's study on the removal of fat and epidermis from excised skin has little effect on the elastic

modulus or strength, an indication that strength rests in the dermis, particularly in the collagen. If the elastic modulus of a piece of skin is divided by its collagen content, the values obtained approximate those of hydrated tendon, which is nearly pure collagen. When the skin is stretched and the slack taken up, the collagen fibers, normally running in various directions become oriented to the force. Once the fibers have become so aligned skin exhibits the tensile properties of collagen alone. The tensile strength of skin, therefore, lies predominantly in its collagen. However, tensile strength is more than collagen content; in wound healing, for example, the tensile strength of an incised wound continues to increase long after the initial rise in collagen content has leveled off. This indicates that the remodeling of the scar can contribute significantly to its tensile strength. Both the amount and the architecture of collagen, then, determine the tensile strength of the dermis. How the dermal collagen network regains its normal organization and returns to the resting state after mechanical distortion is still controversial, this may be the function of the branched, relatively thin elastic fibers.

Viljanto (1971) summarized factors responsible for tensile strength in wound healing. During the first week of healing, neutral-salt soluble collagen and insoluble collagen together with mucopolysaccharides and noncollagenous proteins and some intercellular forces are able to increase the tensile strength to such a level that the edges of most skin

wounds remain in good apposition after removal of the stitches. Thereafter collagen alone is more and more responsible for tensile strength, and the effect of all other factors is restricted to stabilizing of collagen fibrils.

3.3 Fiber orientation

Collagen provides resistance to mechanical stress. The collagen fibrils are laid down dispersed randomly in the ground substance. It is arranged in a loose fibrous network, in the meshes of which are the amorphous ground substance, which provides resistance to compression and bulk flow, elastic fibers, which are thought to restore the morphology of the collagen network after deformation, and reticulin, the function of which is unknown. For the dermis to possess the structural integrity essential for protection, there must be a high degree of physical interaction between its various components; however, for each component to fully express its individual contribution to the tissue a degree of latitude must also be built into these interactions (Montagna & Parakkal, 1974).

Longacre et al (1961) reported that in 85 diffractograms prepared in the Biophysical Laboratory of the Christ Hospital Institute of Research, there are two systems of collagen fibrils in the dermis, which represent a well-organised biologic system, one running parallel to the wrinkle lines and the other perpendicular. The strongest orientation is derived from the collagen fibers parallel to the crease lines. Sections of skin taken from various parts of the

body showed preferential orientation of the collagen in the same direction as the crease lines.

Diffraction patterns obtained from mature linear scars showed orientation of collagen principally in the longitudinal direction of the scar. Diffraction patterns taken of the longitudinal sections of hypertrophic scars showed that the collagen was oriented in the longitudinal extension of the scar. In growing connective tissue and in scar there is the formation of new fibrils and the addition of more fibrils act as templates to extend the polymerisation of collagen. Cross linkage with older fibres increases the strength. Eventually the main process will be the increase in the diameter of the fibres by the accretion of more tropocollagen molecules. The outer layers will be more loosely aggregated and hence more easily extractable.

Reginald & Marlys (1993) described collagen fibrils appear to be cross-striated and form a light and dark pattern. Collagen fibers found in the reticular layer of the dermis are highly undulated fibers and lie in a planar array without orientation. These convoluted fibers are separated from each other by an amorphous ground substance in the relaxed state. Collagen bundles in normal dermis are wavy and contain ample interstitial space. Collagen in burn scar is arranged in a whorl-like pattern and is tightly woven.

Abston (1987) described the change of orientation of collagen fibers with reference to Linare. During the immature phase, the configuration of the

collagen differs between the hypertrophic scar and the nonhypertrophic scar. In the hypertrophic scar, collagen is disoriented, forming whorls, and compact nodules, whereas in the nonhypertrophic scar, it is oriented in parallel bands.

In the semimature phase, vascularity is diminished as the number of fibroblasts; fibrosis becomes more prominent and fibroblastogenesis is diminished. The collagen in hypertrophic scar develops some parallel banding, and the number of nodules and whorls decreases somewhat.

In the mature form, both hypertrophic and nonhypertrophic scar are characterized by a diminished number of capillaries and fibroblasts and increasing fibrosis. The collagen is arranged predominantly in parallel bands.

3.4 Mechanical considerations

The load-transmitting capabilities of the human skin, can be demonstrated through the load-deformation tests on the macroscale carried out in uniaxial tension (Kenedi 1980). Fig. 10 shows the load-deformation test on human skin in vitro.

Fig. 10

The curve shape is concave to the load axis (showing a decrease in deformation with increase in load), and the contractions at right angles to the applied load (the Poisson effect) are comparable in magnitude to the direct extensions.

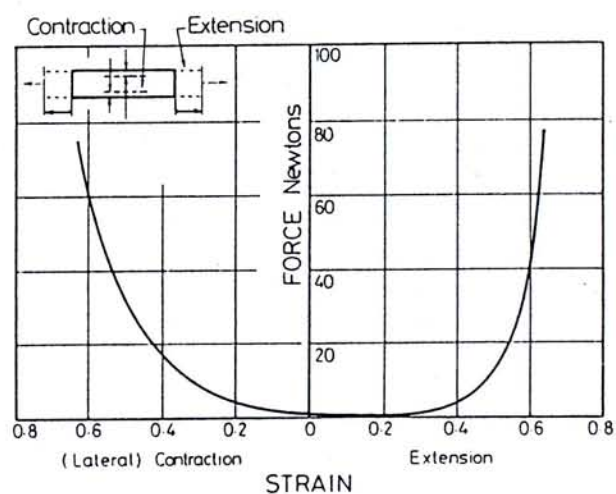


Fig.11 Various human connective tissues in vitro in uniaxial tension.

This curve shows similar load-deformation curves for a number of tissues such as tendon, skin etc.

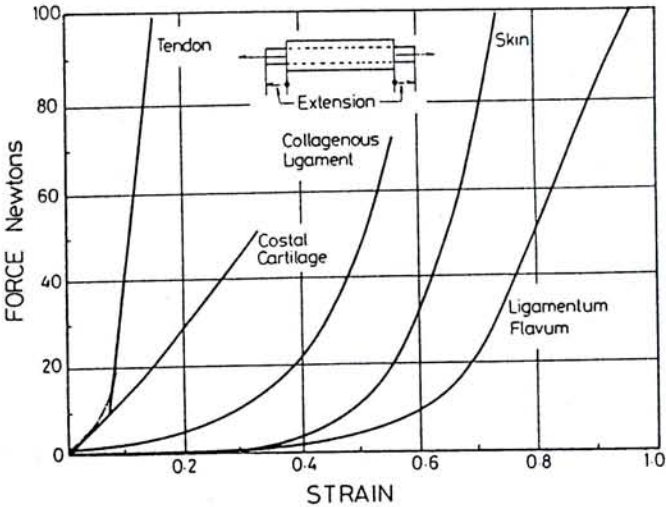
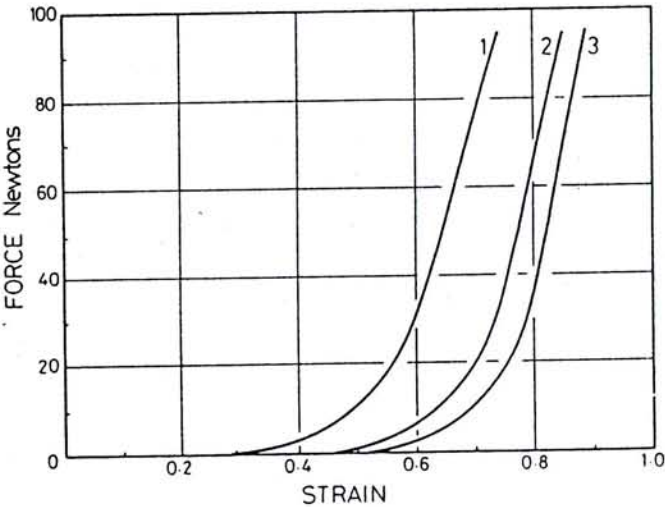


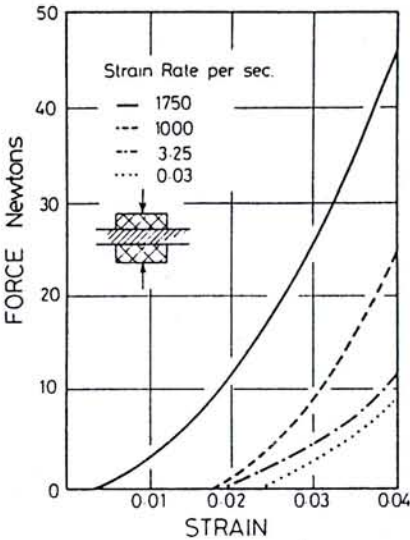
Fig. 12 Human skin in vitro under repeated load cycles 1, 2 and 3.

The figure illustrates the progressive changes consequent on repeated load cycling.



One of the primary addition influences is the time dependence of all such biomechanical characteristics on the macroscale. As an example, uniaxial compression tests on human skin at varying strain-rates show significant time-rate dependence.

Fig. 13
Uniaxial compression tests on human skin in vitro at varying strain rates.



Stress relaxation and creep effects also occur as demonstrated in Fig. 14 (force relaxation in human skin in vitro in uniaxial tension) and Fig. 15 (creep in human skin in vitro under uniaxial tension). On the basis of this it must be emphasized that comparison of test results is only relevant if their time rates of load and strain application are controlled and are known.

Fig. 14 *Force relaxation in human skin in vitro in uniaxial tension.*

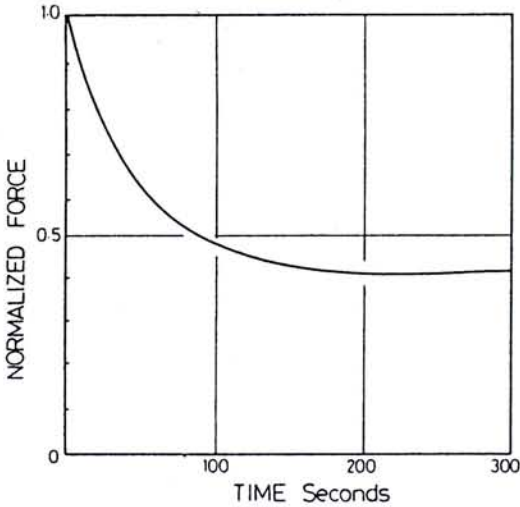
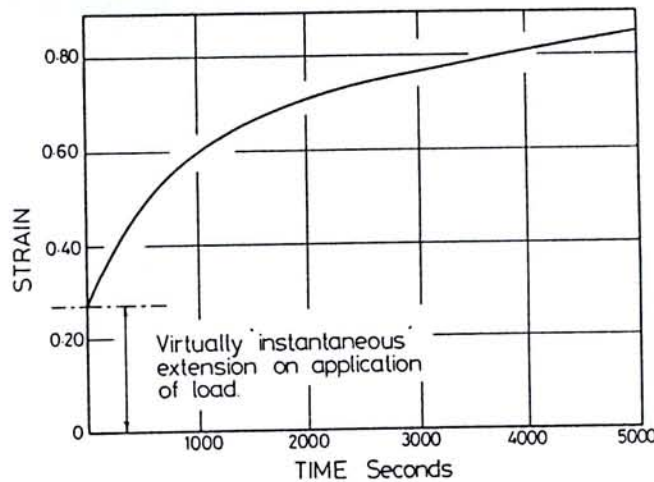


Fig. 15 Creep in human skin in vitro under uniaxial tension.



3.5 Physiological factors

The response of living tissue to the application of load is not solely mechanical. Skin depends for its normal function on factors such as an adequate blood supply. The blood flow within the superficial nutrient capillaries is modified by the application of load and/or by localized deformation with time. Explorations suggest that it is the physical stretch (extension) of the skin which is critical factor - this leads to capillary collapse as the loading and the corresponding skin extension is continued (Kenedi 1980).

Objective studies indicate that a relatively rapid and progressive reduction in blood content of the tissue occurs with increasing deformation. However, there appears to be no specific critical load or deformation at which a sudden collapse of the capillary circulation is manifested, although critical closure of the capillaries can be demonstrated at applied loads relateable to the capillary blood pressure.

While the application of loads sufficient to collapse the capillary circulation for prolonged periods is likely to lead to tissue necrosis, the application of loads of value lower than this but of significant prolonged duration may also produce tissue damage.

3.6 Clinical application

Hypertrophic scarring after thermal injury is described as an elevated inextensible rigid erythemic mass. It consists mainly collagenous connective tissue which is cosmetically un-attractive and often associated with skin and joint contractures. Microstructurally, hypertrophic scars are characterized by an over-proliferation of collagen-forming dense fibrous networks which are mainly responsible for its mechanical properties (Larson et al. 1971, Clarke and Reid 1978).

Low-intensity forces during treatment are usually supplied by special elasticated dressings which are worn by the patients for extended periods of weeks and months. The magnitudes of the pressure applied to the scar surface are dependent on several factors, such as the mechanical properties of the dressing and the tissue. Pressure magnitudes varying between 15-30 mmHg can accelerate the normal maturation process (Naismith and Reid 1978), resulting in flat pale extensible scars which are cosmetically and functionally acceptable. The pressure required to induce such mechanical scar remodeling are uncertain, although it has been suggested that pressures in excess of the mean capillary pressure in normal tissue, usually quoted as 25-30 mmHg are

needed. Similarly, the duration for which these prolonged low-intensity forces should be maintained, appears uncertain, although a remodeling period of at least 6-9 months is generally expected. It has also been suggested that in order to achieve effective remodeling the pressure must be maintained continuously at a constant value during the period of therapy.

4 PHYSIOLOGICAL RESPONSE OF HUMAN SKIN

4.1 Response to mechanical loading

Many types of injurious agents (such as mechanical, thermal or chemical trauma, bacteria and their toxins, viruses and immune reactions) give rise to an inflammatory response in the skin. Inflammatory response is a defensive reaction when the skin performs its protective role as a barrier against the environment. It is normal and frequent. The characteristic signs are immediate and visible including redness, swelling (edema), heat and pain.

4.1.1 triple response

The triple response is an immediate changes take place in inflammation. It is the vascular responses of skin to stimulation by mechanical and other agencies. When a blunt point is used to draw a firm line on the skin. A transitory white line firstly appears which soon becomes a red line; an initial *vasoconstriction* is followed by *vasodilation*. In the third phase, a weal develops which is an area of *local swelling* irregular area of *redness*. The redness is caused by an increased volume of blood flowing through the inflammed area. It has been attributed to the release of some diffusible substance by the injured cells. Injection of histamine produces a similar reaction, but whether histamine or some other vasoactive substance is involved is not known (Ross & Schmid-Schoentein 1991).

There are two divisions of the inflammatory response, vascular and cellular.

The *vascular response* involves vasodilation, and increased vascular permeability gives rise to oedema. It is thought that under the stimulation of the injurious agent, pharmacologically-active will be liberated from the irritated tissues (mast cells) though short live, exert their action on blood vessels. Then follow by small peptides (kinins), mediators of the acute inflammatory response beyond the first half hour.

The *cellular response* involves infiltration of granulocytes and mononuclear cells from the plasma. Normal tissues contain few extravascular polymorphs but in inflammation these cells escape from the microcirculation through the vessel wall. Chemotactic stimuli then 'guide' the cells to the site of injury: if the cells encounter bacteria and other particles then phagocytosis takes place. In this way, invading organisms and tissue debris are cleaned up, preparing the way for wound healing. The phagocytosis of bacteria and other particles can be shown to be greatly enhanced by the presence of antibodies which are believed to coat the bacteria or foreign particles (Wood & Bladon 1985).

4.1.2 reactive hyperemia

Reactive hyperemia is a description of the skin flush due to the restoration of an occluded blood flow. When a hydrostatic pressure is applied to a limb, and the blood flow is occluded for a period of 1 to 5 minutes then restored, the arteriolar and capillary vasodilatation occurred and causing the skin flushed and warm. The simultaneous vasodilatation of the muscular blood vessels induced a dramatic increase in the limb blood flow up to 20 times. The volume of flushing is transient and affected by the duration of the occlusion rather than the magnitude (Al-Rawi 1980, Keele et al 1984).

4.2 Thermal response

4.2.1 Skin temperature

The temperature of the surface of the body varies with the environment, and the body influences skin temperature in such a way as to help maintain the deep or central body temperature constant.

Heat is lost from the body/skin to the environment in several ways :

- by radiation from the body to cooler objects at a distance,
- by conduction and convection to the surrounding atmosphere if its temperature is lower than that of the body,
- by evaporation of water through skin and lung (Keele et al 1984).

4.2.2 Response to heat

Local heat causes local vasodilation, with opening both of the ordinary resistance vessels and of the AV anastomoses. As warmed blood reaches the hypothalamus, reflex vasodilation occurs elsewhere in the skin. The hypothalamic center will also initiate sweating at higher temperatures (Ross & Schmid-Schoenbein 1991).

4.2.3 Response to cold

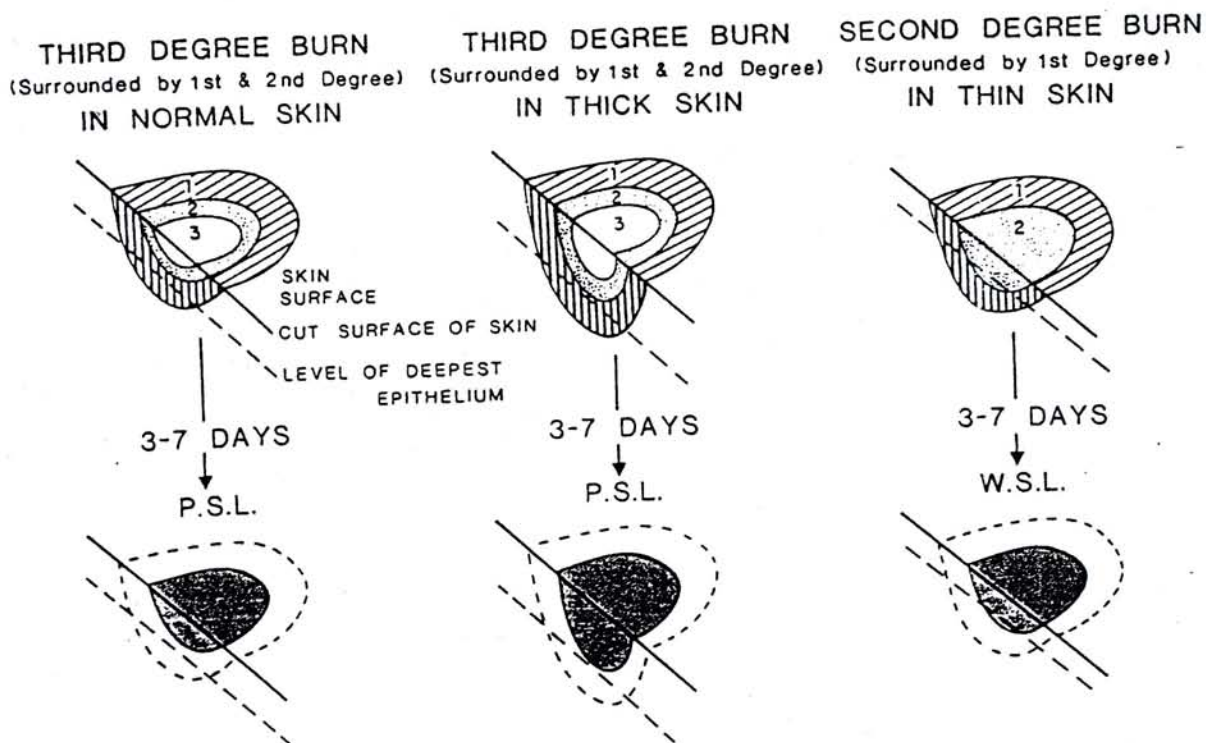
Local cooling of the skin causes local reflex vasoconstriction even in the absence of a change in core temperature (possibly due to increased sensitivity of the vessels to adrenergic nerve impulses, as well as a local cord reflex). Such constriction is reflex and may involve local temperature receptors. With prolonged severe cold exposure, local vasodilation of the skin vessels occurs despite sustained low total skin flow (Ross & Schmid-Schoenbein 1991).

4.3 Local tissue response to burn

Zawacki (1987) reviewed the local tissue response to burn. Burns are four-dimensional that having area, color, depth and they change with time; saucer-shaped with maximum depth of injury located centrally.

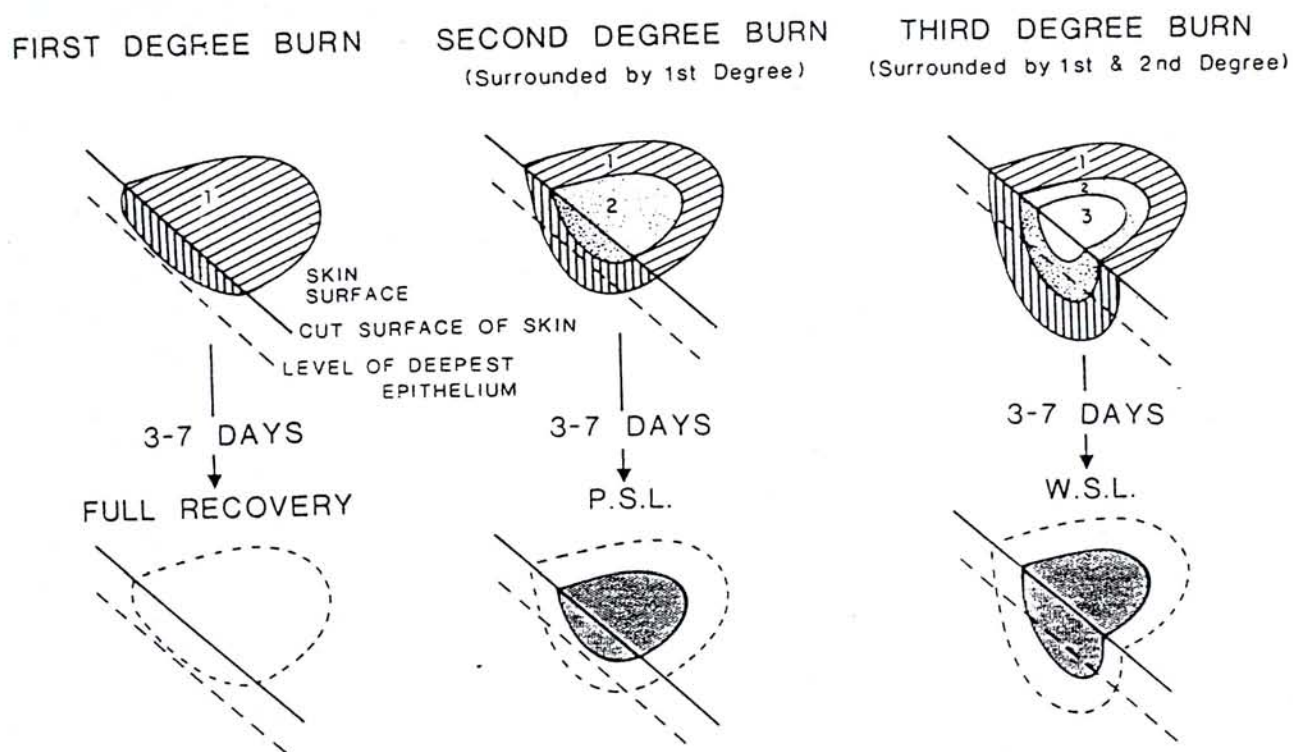
Fig. 16a

Cross-section to show general relation between surface appearance and ultimate depth of necrosis in typical cutaneous burns. Chang WHJ, *The Fundamentals of Plastic and Reconstructive Surgery* 1980, pp.87-108



Examples in which surface appearance (degree) of burn correlates poorly with ultimate depth of necrosis. Even in skin of normal thickness (left fig.), brief high-temperature third degree burns are often PSL in depth. In thick skin (middle fig.) such as the sole, palm, or adult back, third degree burns are almost always PSL in depth. In thin skin (right fig.) such as the pinna in infants, even second degree burn may occasionally be WSL in depth.

Fig. 16b Examples to show the surface appearance(degree) of burn correlates poorly with ultimate depth of necrosis. Chang WHJ, The Fundamentals of Plastic and Reconstructive Surgery 1980, pp.87-108



General relation between surface appearance (degree) and ultimate depth of necrosis (indicated by solid shading) in typical cutaneous burns. Depending on the intensity of burning, burns consist of one, two, or three concentric three-dimensional zones closely corresponding on the skin surface to areas of first, second, or third degree burn respectively: (1) the zone of erythema blanches on pressure and heals in three to seven days; (2) the zone of stasis is initially moist, red, blistered, and blanches but, as usually treated, becomes pale and obviously necrotic in three to seven days; (3) the zone of coagulation is immediately leathery and coagulated, and merges with the necrotic zone of stasis three to seven days postburn.

Character of burn wound

According to Jackson (1984), burn wounds are characterized by the capillary damage which makes the capillaries leak plasma excessively.

The margin of the burn is damaged to a degree which is characterized by *hyperemia* only. it does not blister, it fades in a week, and it may or may not peel or desquamate in 7-10 days.

The center of the typical burn is *coagulated* and usually white. It has no circulation and the capillaries are coagulated in spasm and empty of red cells. It may be wet and letting through plasma as in scalds, or it may be almost dry with the epithelium still adherent as in some burns.

Between the zones of hyperemia and coagulation is a third intensity of burning, the "*zone of stasis*". *It starts with a circulation which then becomes static.* The damaged capillaries in this zone start dilated and leaking, but after minutes or hours, the circulation ceases with the capillaries dilated and stuffed with red cells. It is these red cells that give the zone its mottled pink and white appearance, and responsible for burn edema and shock.

The zone of stasis tissue retains some circulation and this is demonstrated when a few scattered small arterioles bleed on tangential excision after the shock period. Some more evidence show that there will have loss through the retaining circulation. An extensive burn of 60 percent of the surface area will still leak 2-3 liters of exudate a day, this loss can usually be adequately replaced

with oral fluids. 20-40 per cent of the cell mass in extensively burned patients was often lost in the shock period. For example, the red cells being damaged by heat at the time of injury and swept on into the general circulation to be lysed subsequently. Other red cells were broken down into microcytes or spherocytes and were found in the circulation and outside the leaking dermal capillaries.

There is a considerable leak of plasma from the capillaries spreads into the surrounding tissues but reabsorbed. Loss of water vapor is ten times normal through the epidermis; the protein loss in form of exudate account almost 2-3 liters a day from a big wound; and there is also heat loss associated with the loss of protein. The living leukocytes will infiltrate the zones of stasis and wet coagulation and accumulate on the surface . The natural process of autolysis and liquefaction of the burn slough is effected by collagenases form these leukocytes which have invaded it.

Chapter Three BACKGROUND OF THE PRESENT STUDY

This chapter provides the background of the study. The classifications of burn injuries are presented. The content also includes the role of different element in the wound healing process, the aetiology, characteristers, and pathogenesis of hypertrophic scar. Current treatment modalities for hypertrophic scar are also described. Finally, two assessment tools (ultrasonography and elastometry) are introduced for clinical practice.

1 BURN INJURIES AND ITS CLASSIFICATION

The seriousness of thermal injury can be defined by factors such as the extent, depth, location, age, general health and causes.

1.1 Nature/ cause of thermal injury can be classified as :

- Scalds
- Electrical burns : most electrical burns are due to contact with
the electrical supply, lightning
- Chemical burns
- Burns due to hot metal
- Burns due to radioactive emanations
- Flame burns
- Flash burns : usually result from industrial accidents, such as
petrol explosions, power station mishaps and
blowbacks from furnaces

1.2 Depth

According to the circumstances of injury the severity of burning can vary from a simple erythema of the skin to deep charring with involvement of muscle and even bone. The classification of Dupuytren categorized thermal injury into six stages. In most of the first degree superficial burns, tissue destruction involve only the epidermis. There is local pain and erythema without blister formation, and systemic response is absent or mild. Usually no significant injury result and no need for fluid and electrolyte management. Therefore, only second- and third-degree burns are included in calculating the total burn surface area.

Muir et al (1987) compared the pathological classifications of burn injuries in terms of thickness as shown in table 1.

Table 1

(A)	(B) (Scotland, 1942 Department of Health)	(C) (USA)	(D)
Partial thickness skin destruction	1st degree	1st degree	Superficial partial thickness skin destruction
		2nd degree	Deep partial thickness skin destruction
Whole thickness skin destruction	2nd degree	3rd degree	Whole thickness skin destruction

It is believed that the use of the numerical degrees has lead to confusion in the past. (Fig.17 shows the depth of these types of burns in relation to the layers of the skin). Adapted from Muir I.F.K., Barclay T.L. & Settle J.A.D. (1987) Burns and their treatment, 3rd Ed., Butterworths. p.57.

Fig. 17

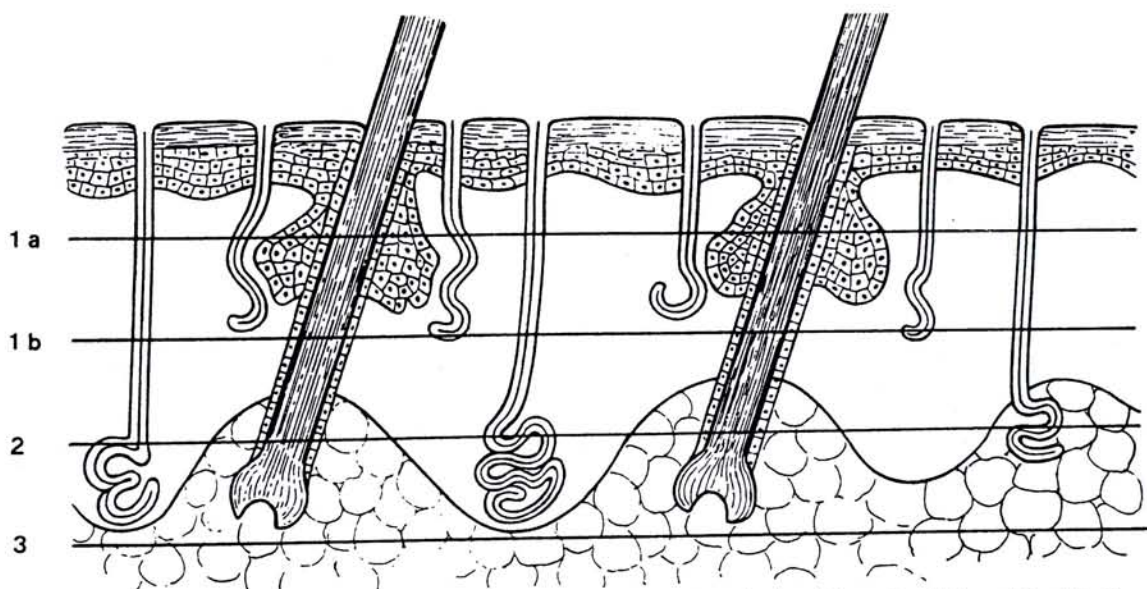


Diagram showing the microstructure of the skin and its relationship to the different depths of burning. Level 1A: superficial partial thickness burn passes through hair follicles, sebaceous glands and sweat glands. Level 1B: also superficial partial thickness burn, but deep to the sebaceous glands. It passes through hair roots and sweat glands. The dermis-fat interface is not breached. Level 2: deep partial thickness burn passes through hair roots and sweat glands, but breaches the dermis-fat interface and cuts across the fat domes. Level 3: full thickness burn passes deep to all epithelial structures.

In order to facilitate the communication, descriptive format will be used for the rest of the thesis.

Superficial partial thickness burns

Superficial partial thickness burns involve only the epidermis and dermis. The wounds appear red and are moist. There is blister formation, and the tactile and pain sensors are intact. The wound will heal in 14 to 21 days with minimal scarring (Warden 1987).

The epidermis is completely destroyed but the hair follicles, sebaceous glands and sweat glands are intact. The dermis-subcutaneous tissue interface does not involve. From the surviving epithelial structures, epithelium rapidly spreads to provide an intact epithelial surface, from which the superficial dead layers flake off revealing a skin which is elastic, supple and of excellent quality, it will be indistinguishable from normal with time (Muir et al 1987).

Deep partial thickness burns

Deep partial thickness burns involve the entire epidermis and dermis, leaving only the skin appendages intact (hair follicles, sebaceous and sweat glands). In contrast to the superficial partial thickness burns, these deeper injuries have a mottled appearance with areas of waxy-white injury. The wound will heal spontaneously in about four to six weeks, with unstable epithelium, late hypertrophic scarring, and marked contracture formation. Early excision and skin coverage is essential (Warden 1987).

The relative numbers and depth of skin appendages varying in different parts of the body. A critical factor is that, because the dermis-subcutaneous interface is not flat, the margin of tissue destruction extends into the subcutaneous tissue at

the places where this pushes up into the dermis (the so-called "fat domes"). This depth of burning can often be recognized at the stage of separation of slough and is sometimes referred to by the alternative name of "deep dermal burn".

Even the small surviving scraps of epithelium suffice for re-epithelialization of the surface, but this is much slower than in the previous type and the healed skin is imperfect and often shows hypertrophic scarring (Muir et al 1987).

Whole thickness burns

Whole thickness burns involve the destruction of the epidermis, dermis, and underlying subcutaneous tissue. The wounds appear white, cherry red, or black and may or may not contain deep blisters. Thrombosed blood vessels may be visible. The elasticity of the burned dermis is destroyed, giving the wound a dry, leathery texture. Marked edema and decreased elasticity may necessitate escharotomy (Warden 1987).

When the full thickness of the skin has been destroyed and there are no surviving epithelial elements, the sequence of events is quite different and the position is much more serious. In the absence of infection, the area of destroyed skin becomes dry, hard and black - the characteristic slough of a full thickness burn.

In the surviving tissue immediately underneath the slough, cellular and capillary activity produces a layer of granulation tissue, and the enzymatic activity of this layer loosens the slough which finally comes away, exposing the red surface of the granulation.

Since the granulation contain no epithelial cells, healing of the area can only occur by ingrowth of cells from the surviving epithelial edge. Initially this is rapid, but as the epithelium grows further and further away from its original site, it grows more and more slowly until finally it stops altogether and occasionally an ungrafted burn may be seen still unhealed even after 15 or 20 years.

While these events are taking place on the surface, changes are also taking place in the granulation tissue. In the deeper layers collagen is laid down. There is reduction in vascularity and finally the tissue becomes a scar tissue. At the same time, the superficial layers of granulation tissue ooze serum which clots and is itself invaded by cells and capillaries to form fresh granulation tissue. As long as the surface remains unepithelialized there is thus a continuous process of laying down of fresh granulation tissue on the surface, while the deeper layers progressively mature into scar tissue.

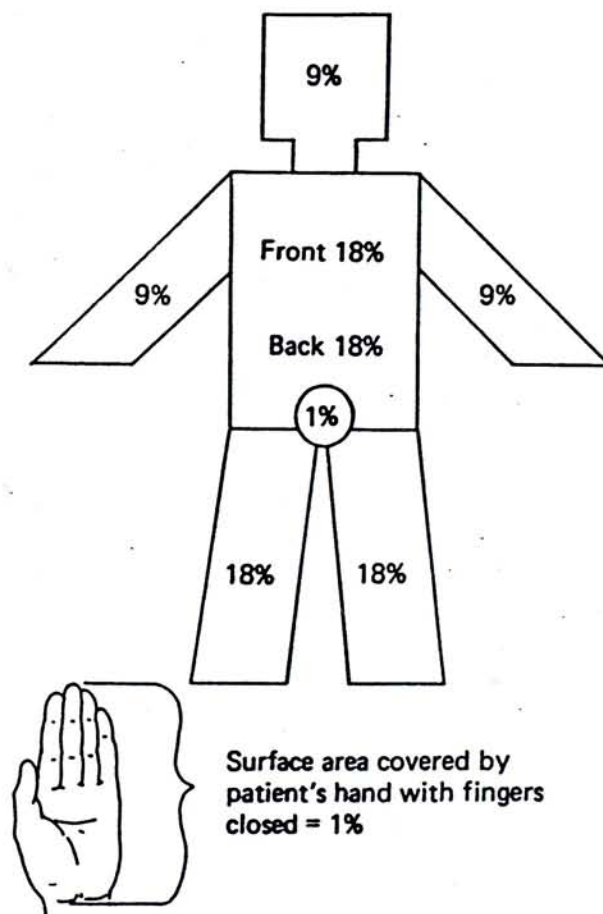
As soon as the scar tissue is formed, it begins to contract with tremendous force, shrinking to only a fraction of its original size. It is this contraction of scar tissue which is the cause of the deformities of severe burns. Surgeons will

use different methods to epithelialize these raw areas by skin grafting, and thus to limit and cut short the formation of scar (Muir et al 1987).

1.3 Extent

The extent of the injury is defined by the total area of burn. Estimation of the total body surface area (TBSA) burned is necessary for immediate fluid replacement and for determining the severity of thermal injury. Even in minor burns, an accurate estimation of the surface area is mandatory (Warden 1987). The Rule of Nine is recommended for the estimation of the surface area involved in individuals more than nine years of age. This does not give as accurate an estimation of the area involved as other formulae, but it is accurate enough for most practical purposes.

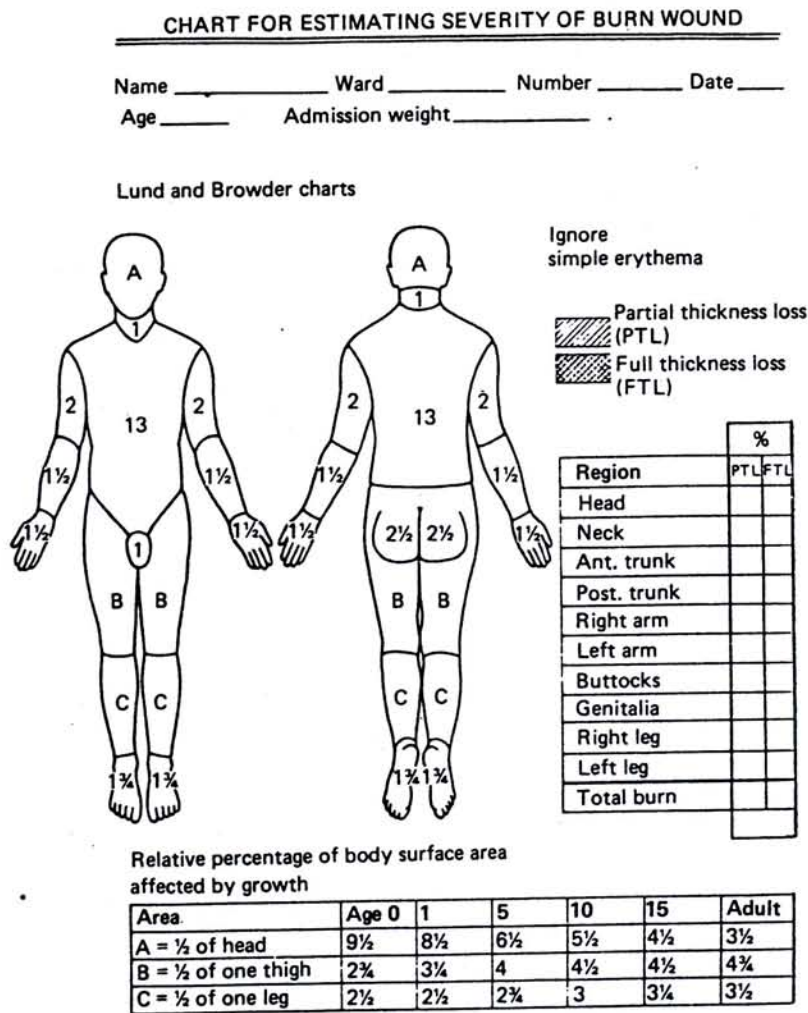
Fig. 18
The Rule of Nine for
estimation of surface area.
Muir et al (1987) p. 29



For smaller areas a good guide is that the area covered by the patient's hand and fingers is 1 percent of the body surface.

It should be remembered that in children the head is a relatively larger proportion and the lower limbs a relatively smaller proportion of the total body area than in adults. For children under 10 years of age, the Lund and Browder chart should be used.

Fig. 19 The modification necessary for different ages. Muir et al (1987) p.30,
Lund and Browder chart for accurate assessment of percentage body surface areas with age being considered.



Lund and Browder chart for accurate assessment of percentage body surface areas

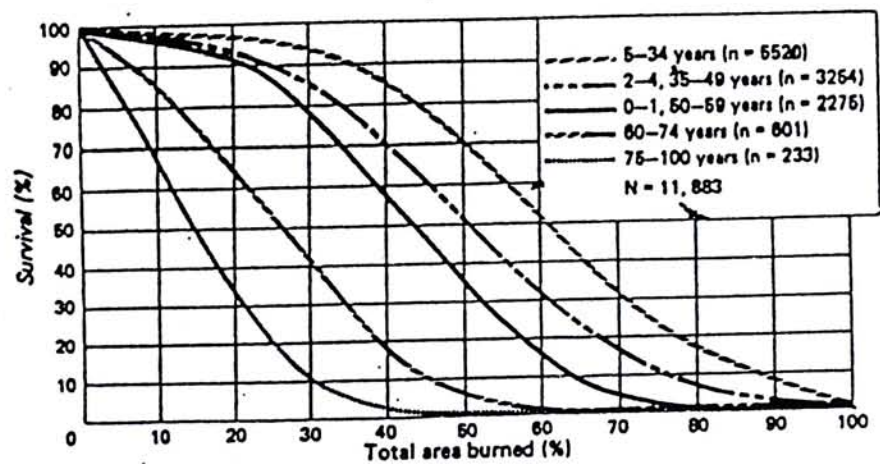
1.4 **Location of the burn**

The eyes, ears, face, hands, feet, and perineum are critical areas that contribute to the severity of the injury.

1.5 **Age**

As much as extent and depth of the burn, age of the victim plays a critical part. Even a relatively small burn in an elderly patient or infant may be fatal, as demonstrated in the figure.

Fig. 20 *Sigmoid curves showing survival of humans as a function of total percentage of body surface burned and age. Feller et al, 1976, JAMA*



1.6 Associated major trauma, inhalation injury

For small burn may be associated with severe inhalation injury, the products of combustion are responsible for lower airway injury. Certain toxic substances from plastic products produce hydrochloric acid and dydrocyanic acid. If the wound is secondary to electrical injury, there may be deep muscle injury associated with a small cutaneous injury. It may also produce cardiac arrhythmias that require immediate attention.

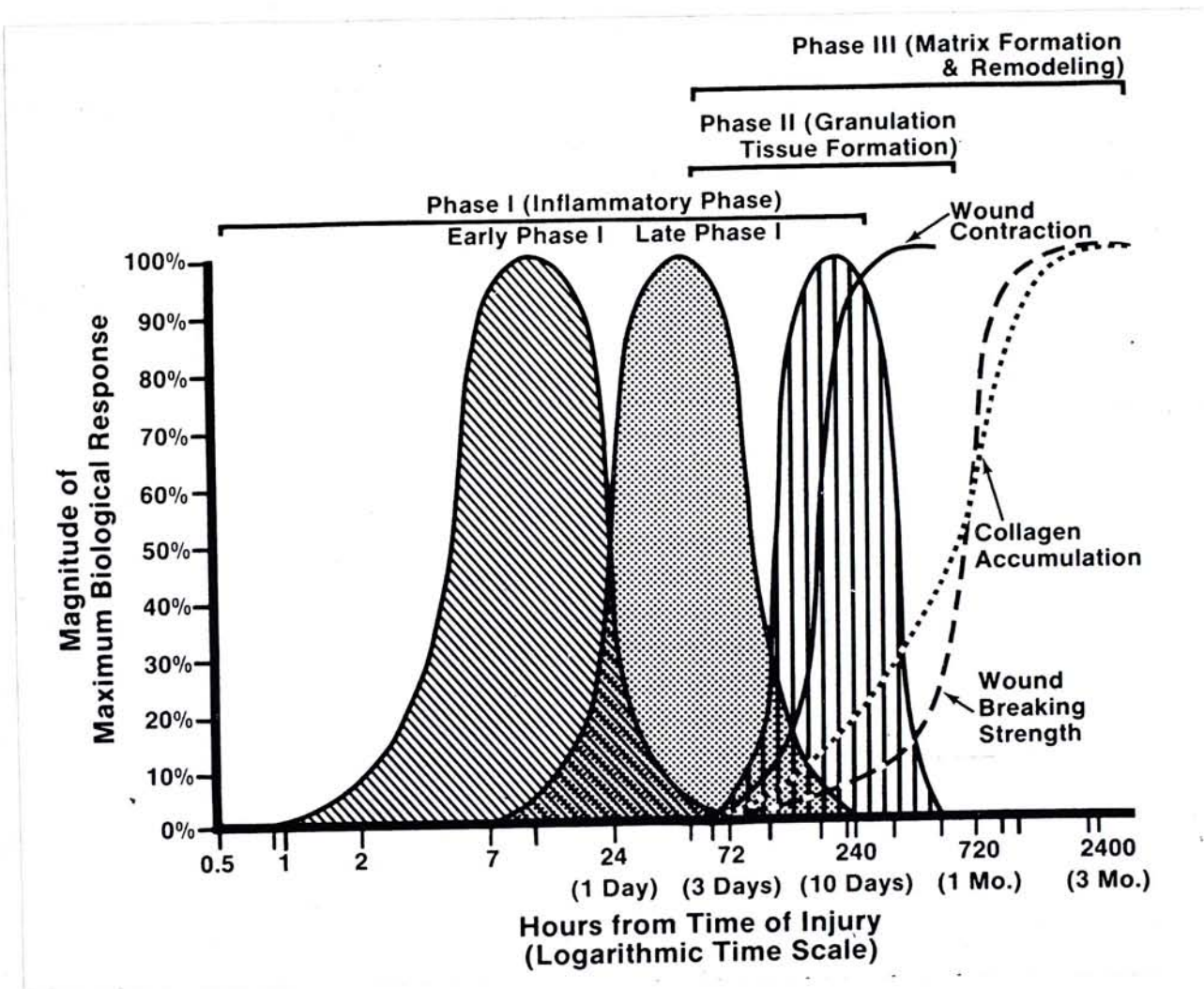
1.7 General health status

Premorbid physical and psychosocial disorders complicate the recovery from burn injury. The disorders include cardiac or pulmonary disease, diabetes, alcoholism, drug abuse, and psychiatric illness.

2 **WOUND HEALING PROCESS**

Wound healing is a combination of vascular responses, cellular and chemotactic activity. The release of chemical mediators within the wounded tissues are inherent, interrelated components of the healing process. There are three overlapping phases : the inflammatory response, reepithelialization and contraction, and finally connective tissue formation. (Fig.21, showing the three overlapping phases of wound repair, from Daly T.J. 1990 Wound Healing: Alternatives in Management, p. 15).

Fig. 21



Inflammatory reaction :

Healing begins with an inflammatory reaction, followed by formation of granulation tissue. Initially, migration of leukocytes into the wound is accompanied by macrophages. New capillaries are generated and a rich capillary network is

established. Fibroblasts migrate from the adventitia and begin to lay down collagen and mucopolysaccharides. With time, the capillaries deep in the wound diminish in size and number. The collagen strands become more compact and are reoriented. As the granulation tissue develops, the eschar is separated. This process takes place (usually) over a three- to six- week period; then autografting is necessary (Abston 1987).

The initial healing reactions to wounding, the vascular and cellular responses, are manifested as the inflammatory response. Local vasodilation, fluid leakage into the extravascular space, and blocking of lymphatic drainage produce three of the four cardinal signs of inflammation including redness, swelling, and heat. The fourth sign, pain, is produced by distention of tissue spaces from swelling and pressure, and by chemical irritation of nociceptor receptors. The primary function of these events is to bring phagocytes to the inflamed area to destroy bacteria and rid the tissue spaces of debris from dead and dying cells so repair processes can begin (Kloth & Miller 1990).

Re-epithelialization and Contraction (the repair phase of wound healing) :

With reference to Daly T.J. (1990), cellular activity reaches a frenzied pace. An integration of inherent biochemical, synthetic, and regenerative processes results in repair of the traumatized tissue. The energy expended in yielding these reparative results must be used in the most efficient manner. This energy-efficient requirement is fulfilled by recreating a permeability barrier (re-epithelialization) and reducing the wound surface area (contraction) to accelerate the wound healing process. A new blood supply (neovascularization) and replacement and reinforcement of the injured

tissue (fibroplasia) are also fundamental processes involved in the repair phase of wound healing.

Wound contraction is the process that closes wounds after loss of tissue. The wound space area is decreased during contraction. This dermal process causes closure of the wound, with or without prior epithelialization. In fact, much of the epithelium is ultimately lost in the fully contracted wound. Contraction involves movement of pre-existing tissue centripetally and not formation of new tissue. Contraction does not always go to completion, except occasionally in small wounds.

Contracture is the end result of wound contraction that may be the result of contraction itself, fibrosis, adhesions, or muscle or other tissue damage. Contractures usually present functional problems in the healed wound.

Remodeling of connective tissue in wound healing :

The end result of contraction is a stable wound with a constant turnover of collagen with remodeling of the matrix. The synthesis and deposition of collagen initially is more random and the continuity of stresses and strains on the tissue causes an alignment of collagen fibers to occur, which strengthens the wound. Wounds that are under stress heal more securely at an earlier date. Allowing patients to move about freely after the initial stages of wound repair are completed seems reasonable. wound contraction then continues into the third phase of wound healing, matrix formation and remodeling.

2.1 Role of collagen in wound healing

Collagen is the principal structural body protein providing strength and stiffness to dermal tissue. There are 5 major types of collagen identified and carry different functions. Only type I and type III are found in dermis and are involved in wound healing. Throughout the healing process, there is constant collagen lysis and synthesis. In the initial phase (4 to 6 weeks) of healing, synthesis occurs more rapidly than lysis (up to 41 times that in normal skin). Then, it is followed by a reverse activity level. The lysis occurs more rapidly than synthesis. An imbalance can occur where collagen lysis cannot accommodate collagen synthesis, resulting in overdeposition of collagen, and hence, development of hypertrophic scar (Price 1990).

2.2 Role of oxygen in wound healing

Oxygen is required to supply the energy for the high metabolic needs of a healing wound and essential for the tensile strength of the wound. Oxygen may be the rate-limiting substrate in many wounds. It is required for the hydroxylation of proline and lysine in collagen synthesis. Oxygen content will affect the growth of fibroblasts. Lack of oxygen will lead to wound breakdown. The tensile strength of wounds has been found to be proportional to the oxygen delivery to a wound (Greenhalgh & Staley 1994).

2.3 Role of fibroblasts and myofibroblasts in wound healing

Baur (1978) suggested that the myofibroblasts played a principal role in the development of hypertrophic scars. The contractility of the myofibroblasts contribute to the contractile nature of wound healing tissue.

Under observation, *myofibroblasts* comprised approximately 50-75% of the total cellular population in the dermis of clinically active hypertrophic scar tissues. The contraction of the myofibroblasts foreshortens the adjacent collagen fibers. The resultant sinusoidal patterns of collagen are then fused into solidified masses by mucopolysaccharide ground substances synthesized by normal fibroblasts and possibly by myofibroblasts. The foreshortened and fused mass then causes an elevation of the overlayered tissue resulting in the inherent rigidity and density so commonly observed in hypertrophic scars. The orientation (long axes) of the myofibroblasts in this case was observed to be parallel to the direction of contraction. This observation likewise implicated the myofibroblasts as the cause of contraction since the other tissue components such as collagen, elastin, vessels, capillaries, etc., were often omidirectional. Thus the only components of the scar tissue uniformly oriented with respect to the direction of contraction were the myofibroblasts. The implications of myofibroblast contraction and synthetic activities in contractures appear similar to the proposed concept of hypertrophic scar formations, the difference lying only in the orientations of the cells (Baur 1978).

In the examination of both active and mature hypertrophic scars with scanning and transmission electron microscopy. Four cell types were identified : the fibroblast, myofibroblast, fibroblast, myofibroblast. The fibroblast and myofibroblast contain intracellular collagen fibers, their role in remodeling the collagen is similar to the role of the osteoclast in the degradation of collagen in bone. The myofibroblast and the myofibroblast contain intracellular contractile bundles. The 'elastic' cells were most prevalent in the mature hypertrophic scars.

2.4 Role of mast cells in wound healing

Kischer et al (1978) reported a finding of increased mast cells in the hypertrophic burn scar, but as it matures, the number of mast cells decreases. The exact role of the mast cell in the hypertrophic scar is unclear.

3 HYPERTROPHIC SCAR

As thermal injury always result in hypertrophic scar, this part of the thesis will study the hypertrophic scar from various aspects :

3.1 Etiology factors

The degree of scarring left by a burn (Muir 1987) depends on the depth and site of the burn and the age of the patient.

However, Deith et al (1983) studied 245 burn areas of 100 patients with different age, races and gender. Several risk factors contributed to post-burn hypertrophic scar were analyzed. The result shown that

3.1.1 Age

There is no correlation between patient *age* and the development of wound problems was found.

3.1.2 Time for wound healing

It was the most important factor in predicting which patients would develop burn wound problems. If the burn wound healed between 14 and 21 days then one third of the anatomic sites developed wound problems, while if the burn wound healed after 21 days then 78% of the burn sites were hypertrophic.

3.1.3 Racial factor

Race was a more important factor than age in predicting which patients would develop elevated burn scars, but less sensitive than predicting by wound healing time.

3.1.4 Depth of injury

Depth of injury was considered as an important factor since burn involve the reticulodermis are prone to heal with hypertrophic scar while more superficial burns which involve the papillary dermis frequently heal without scarring, although pigmentation change may occur. Muir et al (1987) held similar opinion, in superficial partial thickness burns, the healed skin is of good texture and elasticity; at first there may be some depigmentation of the area, but this gradually returns to normal. All deep partial thickness burns result in scarring, and in full thickness burns scars form at the margins of grafts and in areas which have healed by secondary epithelialization. Burns scars, like all other scars, go through a series of phases, being initially flat and fairly inconspicuous, then red, thick and hard, and finally flat, white and soft.

3.1.5 Location

The location of the wound has certain implication. There was a higher incidence of hypertrophic scars in the chest, back, shoulders, and buttocks where the wound tension was the greatest.

3.2 Characteristics

3.2.1 Histologic characteristics

The development of hypertrophic scar is suggested by Linares et al (1972), in three stages : immature, semimature, and mature (Abston 1987).

Immature stage :

The configuration of the collagen differs between the hypertrophic scar and normal scar. In the hypertrophic scar, it is disoriented, forming whorls and compact nodules, whereas in the nonhypertrophic scar, it is oriented in parallel bands.

Semimature stage :

Vascularity and number of fibroblasts are diminished in both scar. Fibrosis becomes more prominent and fibrillogenesis is diminished. The collagen in hypertrophic scar develops some parallel banding, and the number of nodules and whorls decreases somewhat.

Mature stage :

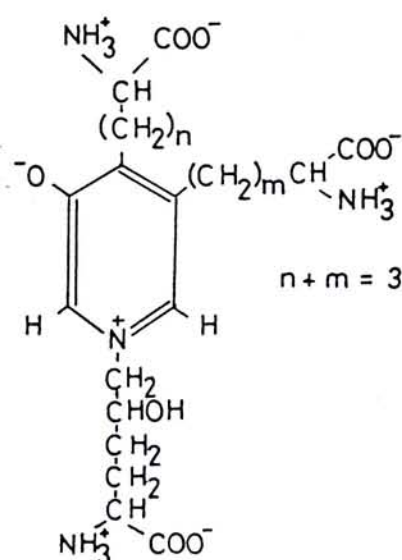
Both hypertrophic and mature scars are characterized by diminished number of capillaries and fibroblasts and increasing fibrosis. The collagen is arranged predominantly in parallel bands.

The collagen filaments or fibrils are smaller in diameter in hypertrophic scars than in normal dermis, and the interstitium contains more filamentous material than the interstitium of normal dermis. In mature,

nonhypertrophic scar, the collagen filaments are larger than in hypertrophic scar, but not as large as in normal dermis. Electron microscopic examination of the collagen filaments in cross-section demonstrates a diameter size in nodular areas of hypertrophic scars. With reference to Linare (1973), in normal skin the diameter range from 400 Å to 1200 Å, with the predominant diameter about 1000 Å. For whorl area, diameter ranges from 300 Å to 500 Å for filaments.

It is conceivable that intramolecular and intermolecular crosslinks of collagen may be involved in the formation of hypertrophic scar. A new crosslinking amino acid was isolated namely *pyridinoline* from collagen by Moriguchi & Fujimoto (1979). An appreciable amount of pyridinoline was found in collagen in hypertrophic scar, low content could be detected in mature scar, but virtually absent in collagen of normal.

Fig. 22
Chemical structure
of pyridinoline



3.2.2 Biochemical Character

During the hypertrophic phase, synthesis of collagen and connective tissue is increased for at least two years after wound closure. The process of collagen degradation in human scar is not fully understood and not all of the activators of synthesis and degradation have been identified. With maturation, the hypertrophic scar softens, thins, and flattens, and synthesis and degradation are balanced.

Hoopes et al (1971) suggested in the initial reaction in the crosslinking of collagen in hypertrophic scars, enzyme lysyl oxidase (the catalyst in the formation of ϵ aldehydes), increased sharply two to three months after the occurred, remained high for two to three years, and then slowly decreased to normal about five years after injury.

The activity of enzyme prolyl hydroxylase (catalyze the hydroxylation of peptide-bound proline to hydroxyproline during the synthesis of chains of collagen peptides) were corrected with the rate of collagen synthesis, and its activity in granulation tissue was 25 to 50 times that in normal skin. Following healing or grafting, the activity of prolyl hydroxylase decreased but remained elevated for two years - from 5 to 20 times normal. After a total of four years, however, it was below that of normal skin.

Study of collagen solubility confirmed there is an increase quantities of salt-soluble collagen. Concentration of insoluble collagen in hypertrophic scar was decreased initially in comparison to normal skin; it was greater than in normal skin later, but the concentration of salt-soluble collagen decreased to normal. The transformation of soluble collagen to insoluble takes place by formation of covalent crosslinks, and the increase in insoluble collagen after two years correlates with the findings of increased activity of the lysyl hydroxylase during the first two years after the injury.

Ratio of Type III to Type I collagen is increased in granulation tissue and hypertrophic scars more than twice that in normal skin. This increase in the quantity of Type III persists for two to three years after the injury.

Pyridinoline is identified as an amino acid serves as a crosslink of collagen. According to the finding of Moriguchi & Fujimoto (1979), pyridinoline is another example of the abnormal nature of collagen in hypertrophic scar. It has been reported that the collagen produced after an injury of human skin is initially stabilized by a crosslink derived from hydroxyallysine. In normal wound healing, there is a changeover with time to the crosslink derived from allysine. In contrast, hypertrophic scar fails to follow such the time-related changes of normal skin. Since pyridinoline is considered to be derived from hydroxylysine and

hydroxyallysine residues, it is understandable that it is formed in hypertrophic scar.

3.2.3 Mechanical properties

Clark et al (1987) monitored the progress of post-burn hypertrophic scar by measuring the mechanical properties, the modulus of elasticity (E) and strain (ϵ) at different load intensities.

Clinically, scar tissue were graded as the following :

<u>Grade</u>	<u>Scar assessment</u>
0	Normal skin
1	Soft, paper thin, pink
2	Soft, thin, pink to red
3	Firm, moderate thickness, red
4	Firm, thick, red to purple
5	Hard, thick, purple

Correlation of clinical assessment of hypertrophic scar grading has been achieved with these mechanical properties. Higher scar grading is indicative of increased stiffness (Modulus of Elasticity) and decreased extensibility.

According to Stark (1977), the load-extension curves for normal skin display an initial compliant phase, during which a large extension was

produced by applying a low load, followed by a progressive stiffening with increased extension. The time-dependent initial behaviour period is associated with the response of the elastin content of the skin whereas the stiffening is a result of the collagen content resisting stretching after an initial realignment phase of these fibres.

In comparison with Stark's studied of behaviour of normal skin, Clark et al (1987) found that even when scarring tissue subject to the smallest loads (less than 20gcm^{-1} tab width) it stiffens abruptly displaying a very different response to that of normal skin.

3.3 Pathogenesis of hypertrophic scar

An immature scar has a disorganized, random pattern and a mature scar has a more linear, organized appearance. Scanning electron microscopy has revealed a dramatic change in the orientation of scar tissue following scar maturation and pressure therapy.

Achaner (1991) summarized how hypertrophic scars differ from mature scars in several ways such as blood flow, tissue gas, filamentous material, mast cells, chondroitin sulfate and level of enzyme proline hydroxylase.

3.3.1 Blood flow

Studies utilizing the laser Doppler fluorometry and capillary microscopy have demonstrated increased blood flow.

Kischer et al (1982) studied the microvessels in normal skin, granulation tissue, hypertrophic scar, keloid, and mature scar by transmission electron microscopy. Comparative observations suggested that most microvessels in hypertrophic scar and keloid are occluded or partially occluded, apparently owing to an excess of endothelial cells. Endothelial cell contraction was observed in hypertrophic scars and keloids. Among findings from statistical analyses were that the patency of microvessels in hypertrophic scar is significantly less than that of microvessels in normal skin. Endothelial cell density is greater in nonpatent vessels than in patent vessels. The observed extent of microvascular occlusion supports the theory that hypoxia is involved in the generation of hypertrophic scar.

3.3.2 Tissue gas

Tissue gas studies in hypertrophic burn scars have demonstrated a decrease in oxygen and an increase in carbon dioxide compared with control sites.

3.3.3 Filamentous material

Smaller collagen filaments and more filamentous material are found in hypertrophic scars.

Kischer & Brody (1981) studied the structure of the collagen nodule from hypertrophic scars. The collagen nodule is the structural unit of all hypertrophic scars. Nodules are composed of a marked increase of unidirectional collagen fibrils aligned in a highly stressed orientation. There is also a marked increase in the number of fibroblasts. Few microvessels appear within the nodule; rather, they remain peripheral and encompass the main body of the nodule as a net. There is a heavier concentration of microvessels (3-13 micrometer in diameter) at the terminal ends, many of which are occluded. The occlusion appears due principally to an apparent increase in the number of endothelial cells. The character of the nodule and its relationship with a peripheral semiocluded microvascular network suggests an origin of the hypertrophic scar to be related to revascularization of a deep wound.

3.3.4 Mast cells

Mast cells are more prevalent in hypertrophic scars but decrease as the scar matures. Although the role of the mast cell is still unclear for over a decade, this appearance does correspond with the period of intense pruritis.

Kischer & et al (1978) analysed the mast cell activity. They tried to quantitate the mast cells in wounds and subsequent healing stages. Observations were made on several cases of hypertrophic scarring treated with mechanical pressure for accelerated resolution and early maturation of the scar. Some indications were drawn :

- the hypertrophic scar contains significantly greater numbers of mast cells than the other tissues studied,
- as granulation tissue develops interstitial collagen, mast cells begin to appear,
- mature scars contain significantly fewer mast cells than hypertrophic scars,
- based on mast cell data, the effect of pressure therapy is first detected in the upper and middle reticularis of the dermis, and
- on a mast cell statistical basis mature scar and hypertrophic scar under pressure are indistinguishable.

Kischer et al suggested that the magnitude of mast cells reflects the stage of healing from deep surface injury. The greater number of mast cells occurs in hypertrophic scarring stage and is reduced as maturation or resolution occurs.

Hypertrophic scar is regarded as a stage of healing because clinically all the mature scars had previously passed through a period of

hypertrophic scarring. Further, scars from normal healing are indistinguishable histologically and ultrastructurally from mature scars resolved from a previous period of hypertrophy.

The same study in 1978 further indicated that the effects of pressure on numbers of mast cells may be identified in the papillary dermis, upper, and middle reticularis, since, under pressure these areas have similar mast cell counts as mature scars. However, the lower reticularis is not similar in mast cell counts to the corresponding area of mature scars. This suggests that the effects of pressure therapy are not realized at this depth until a much later time, perhaps the time at which constant pressure is no longer necessary, and final permanent resolution of the hypertrophic scar has taken place.

3.3.5 Chondroitin sulfate

Sulfated mucopolysaccharides have been found in mast cells it seems doubtful the amount would be enough to account for the 14-fold increase of chondroitin sulfate A in hypertrophic scars over that in normal skin. However, it should be significant that the levels of mast cells in hypertrophic scars are maintained throughout the period of hypertrophy, which often is years. This suggests a supportive or complementary role of the mast cell for hypertrophic scarring. Such an intimate relationship is further supported by the significant reduction of

mast cells under pressure treatment, which mechanically resolves the hypertrophic scar.

3.3.6 Enzyme proline hydroxylase

Level of the enzyme proline hydroxylase correlate with scar immaturity, rate of collagen synthesis, and presence of granulation tissue. The presence of granulation tissue has been deemed favorable for wound healing. The formation of capillaries and the subsequent proliferation of fibroblasts probably lead to hypertrophic scars.

The rates of collagen biosynthesis and the tissue concentrations of collagen in normal scar, hypertrophic scar were determined as a function of the duration of the lesions.

Craig et al (1975) examined the *rate of collagen synthesis with respect to the time elapsed after wounding*. Normal scar synthesized collagen at a relatively constant rate, in both hypertrophic scar and keloid the rate of collagen synthesis during the first 2-3 years after wounding was approximately twice as high as in normal scar and subsequently fell to the level found in normal scar.

The tissue concentration of collagen in normal scar was relatively constant with respect to the time elapsed after wounding, whereas in both abnormal types of scar there appeared to be a continuous increase up to at least 4 years.

In both hypertrophic scar and keloid, the pattern of changes in the rates of synthesis and in the tissue concentrations of collagen are very similar. These observations may indicate that the events leading to the pathogenesis of hypertrophic scars and keloids are essentially similar.

In normal scar it is apparent that the rates of synthesis and degradation of collagen are in equilibrium since there is no excessive accumulation of collagen in either case. Initially the rate of collagen synthesis in hypertrophic scar and keloid is twice as high as in normal, but 2 or 3 years later falls to the same level as in normal scar. Despite this fall in rate of collagen synthesis there is a continued excessive deposition of collagen, which suggests that there is also deficiency in the mechanism of collagen degradation in the two abnormal tissues.

The observations raise the possibility that there may be two phases in the production of hypertrophic scar and keloid, the first being characterized by abnormally high rates of collagen synthesis and possibly abnormal degradation of collagen, and the second being characterized by essentially normal rates of collagen synthesis and presumably decreased degradation.

Hoopes et al (1971) reported the enzyme activities in the epithelial cells were increased slightly in both hypertrophic scars and keloids. Greater *enzymatic changes* were observed in the dermis than in epithelium. The most striking increase (9 to 13 times normal) was found in the glucose 6-phosphate

dehydrogenase activity in the dermis. the enzymes of amino acid and fatty acid metabolism were decreased in the dermis. the dermal DNA content was increased two times normal in hypertrophic scars and in keloids: this may imply an increase in cell population of fibroblasts. The epidermal DNA content exhibited no significant change.

Kischer (1982) proposed a pathological mechanism of the development of hypertrophic scars. He suggested that this mechanism arisen from the inflammation associated with the *deposition of fibrin within the matrix of the granulation tissue*. This process may give rise the progressive occlusion of developing blood vessels which in turn gives rise to local hypoxia. This results in excessive deposition of fibrous tissue. The deposition of fibrous tissue probably follows the alignment of the polymerized fibrin thus giving rise to the characteristic hypertrophic scar 'nodule'. Eventually the process becomes self-limiting as the hypoxia progresses to a point which results in cell death within the 'nodule'. Release of hydrolytic enzymes from the dead cells causes breakdown of fibrous tissue which allows restoration of blood vessel patency. Hypoxia is alleviated and there is a gradual reduction in cell numbers and activity within the affected area. Scar resolution, often accompanied by contraction, continues until a stable mature scar is formed. There is wide variation in the duration of these processes. Some scars may resolve in a matter of months whereas others may still be active after several years. This mechanism is supported by some clinical observation, such as : active scars are apparently hyperaemic and usually inflamed and pruritic; considerable

abnormality of structure of blood vessels was found (Page et al 1983) within the scar tissue, defective sympathetic innervation of the regenerating blood vessels was noticed in the early stages of development of the scar, even in matured scars the structure of blood vessels was markedly abnormal.

Shakespeare & Renterghem (1985) used the scanning electron microscopy and found that the hypertrophic scar show a completely different structure of collagen at the *epidermis-dermis interface* when compared to normal skin. When the hypertrophic scar matured the interface was remodeled to resemble more closely the interface observed in normal skin. The secure and stable attachment of epidermal cells to the underlying fibrous tissue may therefore alleviate the inflammatory response associated with fibrin deposition in the developing scar. The process of wound contraction may be important in remodeling of the hypertrophic scar fibrous tissue surface until it comes to resemble more closely the surface of collagen in normal skin and thus, possibly, becomes a more receptive surface for the anchorage of epidermis. So, the attachment of epidermal cells to the surface of the fibrous tissue developing in the burn wound may be an important aspect of the pathogenesis of hypertrophic scarring.

3.4 Histopathology

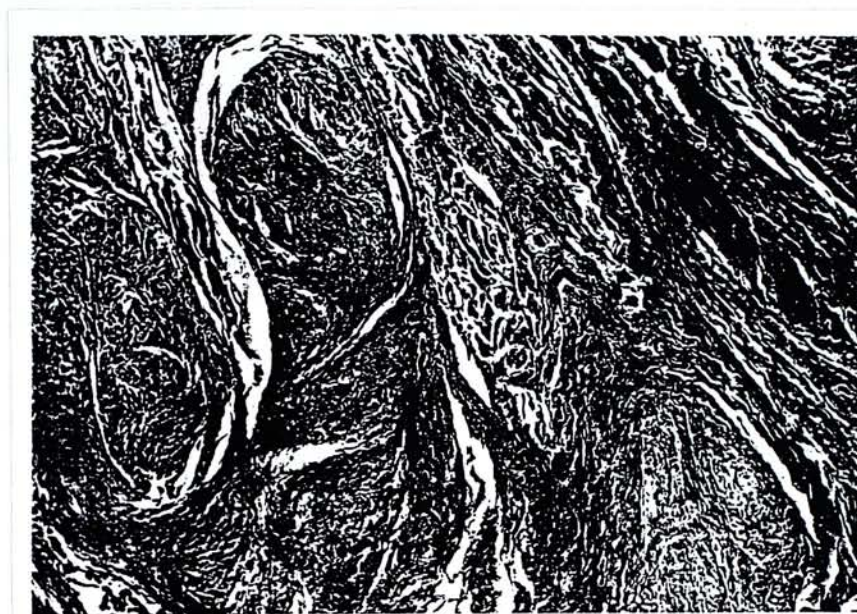
Lever & Lever (1983) stated that keloid and hypertrophic scars are indistinguishable from one another on histologic examination, since both show formation of whorls and nodules. Whereas the whorls and nodules persist in

keloid, they ultimately flatten out in hypertrophic scars. The length of time over which new collagen is formed, and the arrangement of the newly formed collagen contribute to the difference between normal wound healing and healing with a hypertrophic scar or keloid.

Normal wound healing proceeds through an early inflammatory stage to a 'fibroblastic' stage in which one finds granulation tissue composed of numerous capillaries, fibroblasts, and collagen fibers. The collagen fibers in the reticular dermis show a parallel, wavy orientation. Usually after 5 weeks, the number of capillaries and fibroblasts has decreased, and the collagen lies as thick hyalinlike bundles in parallel arrangement.

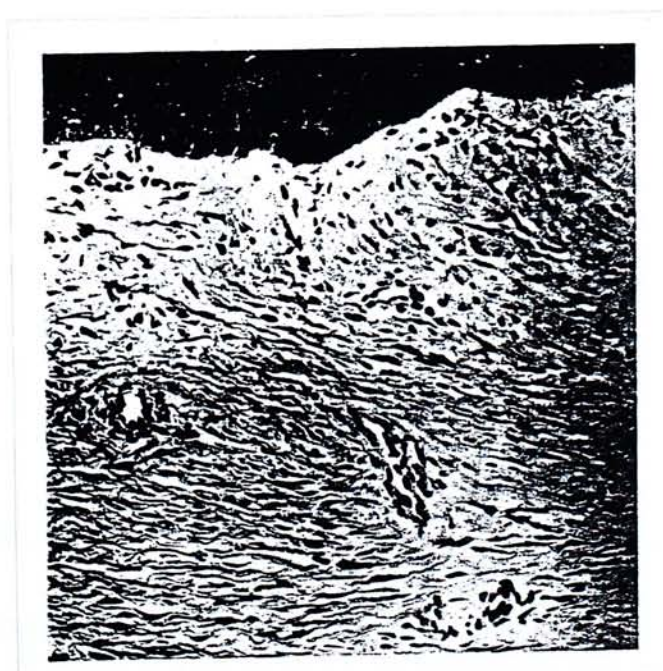
In hypertrophic scars and keloids, the formation of new collagen following the inflammatory stage extends over a much longer period of time than in normally healing wounds. Even in the early period of the fibroblastic stage, one can already see that the collagen fibers in the granulation tissue are arranged in a whorllike or nodular pattern. The nodules gradually increase in size and ultimately show thick, highly compacted, hyalinized bands of collagen lying in a concentric arrangement. Fig. 23 shows compacted hyalinized collagen present in nodular formation. Depending upon whether the nodular condensation of the collagen encroaches upon the papillary dermis, the epidermis appears either flattened or normal.

Fig. 23 Compacted collagen present in nodular formation.



In keloid, the nodular condensation of the collagen persists indefinitely; in contrast, in hypertrophic scars, the thick and hyalinized collagen bundles in the nodules gradually become thinner and straighten out, so that the orientation of the collagen bundles begins to parallel the free surface of the skin. Fig. 24 shows nodular arrangement of hypertrophic scar.

Fig. 24



Under observation, *myofibroblasts* comprised approximately 50-75% of the total cellular population in the dermis of clinically active hypertrophic scar tissues. The contraction of the myofibroblasts foreshortens the adjacent collagen fibers. The resultant sinusoidal patterns of collagen are then fused into solidified masses by mucopolysaccharide ground substances synthesized by normal fibroblasts and possibly by myofibroblasts. The foreshortened and fused mass then causes an elevation of the overlayered tissue resulting in the inherent rigidity and density so commonly observed in hypertrophic scars. The orientation (long axes) of the myofibroblasts in this case was observed to be parallel to the direction of contraction. This observation likewise implicated the myofibroblasts as the cause of contraction since the other tissue components such as collagen, elastin, vessels, capillaries, etc., were often omidirectional. Thus the only components of the scar tissue uniformly oriented with respect to the direction of contraction were the myofibroblasts. The implications of myofibroblast contraction and synthetic activities in contractures appear similar to the proposed concept of hypertrophic scar formations, the difference lying only in the orientations of the cells (Baur 1978).

In the examination of both active and mature hypertrophic scars with scanning and transmission electron microscopy. Four cell types were identified : the *fibroblast*, *myofibroblast*, *fibroclast*, *myofibroclast*. The fibroclast and myofibroclast contain intracellular collagen fibers, their role in remodeling the collagen is similar to the role of the osteoclast in the degradation of collagen in

bone. The myofibroblast and the myofibroblast contain intracellular contractile bundles. The 'elastic' cells were most prevalent in the mature hypertrophic scars.

Kischer (1979) studied the fine structure of granulation tissues. Fibrin was found in all of the granulations from 13 days to 6 ¹/₂ months postinjury. It was seen intraluminally and interstitially. Fibroblast type cells demonstrate morphology which may be predominantly vesicular, granular, or organeeler. Myofibroblasts are abundant and virtually all exhibit microtendons. The significance of the persistent and pervasive fibrin in a deep injury to probable developmetn of subsequetn hypertrophic scarring may lie in promoting endothelial cell proliferation which would occlude the microvessels. Occlusion would then produce hypoxia which, in turn, would stimulate excessive fibroblast proliferation and overproduction of collagen and certain mucopolysaccharides.

3.5 Response towards pressure

Scar tissue consists of young actively growing cells, which readily *respond to changes in stress and external pressure*. Since the scar tissue is responsive in its early stages, it will react favorably to appropriate corrective measures such as exercise, traction, and pressure. As the scar matures it becomes less responsive to therapy; thus the physician should see and evaluate the patient at frequent intervals during the first 6 months after healing (Larson et al 1971).

Serial biopsies of early healed wounds reveals a considerable change in the *collagen*. At first the collagen appears to be in small bundles in an irregular or wavy pattern, as if another force were causing it to "buckle". This gradually continues with the thin bundles becoming thicker and assuming a whorl or nodular formation. Therefore the collagen fibers must play a passive role initially in the development of contractures.

Fibroblasts initiate contraction, resulting first in "buckling" of the collagen and later nodular formation. With traction on the early scar, the fibroblasts elongate and the collagen assumes a more parallel arrangement similar to a mature scar. Later, however, the collagen apparently develops intermolecular cross-linking, i.e., at the fiber level rather than the molecular level, resulting in a more stable and resistant scar.

Early scars are highly vascularized, and the dense connective tissue nodules appear to form near *blood vessels*. Mild traction and pressure cause blanching of the scar with decreased vascularity. Constant pressure or traction may control the blood supply and decrease the unbalanced equilibrium between collagen synthesis and collagen degradation about the young capillaries.

4 TREATMENT OF POST-BURN HYPERTROPHIC SCAR, AND THEIR RESPONSE

Available methods of treatment include surgical excision, pressure therapy, low-dose radiotherapy, topical silicone gel and intralesional injection of steroids.

4.1 Surgery

A removal of the keloid and hypertrophic by atraumatical excision and close precisely with a minimum amount of foreign material and without dead space or hematoma resulting is able to convert the scar into a fine line scar (Ketchum & Cohen, 1974). If undue tension exists, this must be corrected surgically by an appropriate skin graft, flap, or Z-plasty. However, recurrence of the hypertrophic scar is noted. Hence, sometimes, adjunctive measures are necessary.

Longacre et al (1976) held similar opinion. He suggested Z-plasty, application of skin grafts, to a surface defect will result in a decrease in the production of hypertrophic scar. By matching the principal direction of the collagen molecules in the graft to their normal direction in the recipient site, a decrease in the amount of scar tissue production and consequent contracture was noted. Since a great increase in the excretion of polypeptides I and II, proline and hydroxyproline. This was concurrent with the softening and thinning of the scar. This would indicate a change in the balance of collagen metabolism with the resulting resorption of collagen in the scar-infiltrated tissue by the disaggregation process. This is followed by the elimination of the outer layers

of the more loosely aggregated and hence more easily extractable polymers, polypeptides I and II, proline and hydroxyproline. The concurrent processes of production and resorption of collagen allow for the remodeling and revision of the scarred tissues.

4.2 Radiotherapy

Application of radium or Finsen light may bring about absorption in some instances but the process is slow. Ketchum & Cohen (1974) argued that although radiation might be hazardous, evidence of radiation in adjunct with surgery, pressure and chemotherapy was effective. However, routine use of radiation was not recommended.

4.3 Ultrasonic

The literature is sparse regarding the use of ultrasound in the treatment of hypertrophic scars or keloids. One study noted good results with two patients in flattening keloids using continuous ultrasound at $0.8\text{W}/\text{cm}^2$ for 4 minutes. Neither the number nor the frequency of treatments was noted (Staley & Richard 1994).

4.4 Chemotherapy/ Intralesional injection of steroid

Direct application of steroid over a prolonged period of time was reported to produce thinning of the dermis, limited to the area of treatment.

Harvey Kemble (1976) studied the effect of steroid injection therapy. The steroid-treated hypertrophic scar showed a dense homogeneous arrangement of collagen, with small fiber size, that is differed from the active hypertrophic scar only in a relative increase in interstitial space. The appearance is intermediate between active hypertrophic scar and mature scar. Steroid affect fibroblasts, collagen and interstitial mucopolysaccharides. The electron-microscopic appearances of hypertrophic scar show little extrafibrillar & extracellular space, but significantly more in the maturing steroid-treated scar. This is probably a reflection of the diminished fibroblast production of collagen, as well as an increase in collagen lysis.

Ketchum & Cohen (1974) suggested a protocol of using triamcinolone in the treatment of scars. For scars less than 15 cm², inject only. For lesion larger than 15 cm², excision and resurfacing with a split-skin graft is recommended, with injection of the wound edges at the time of surgery. Since injection therapy alone would be too time-consuming for these larger lesions. Ketchum & Cohen also reported the evidence that triamcinolone might act by enhancing collagen degradation. When injected with triamcinolone, scars exhibited increasing amounts of soluble collagen. This increase in soluble collagen probably represented an increased collagen degradation. Intralesional

injections of triamcinolone do not inhibit collagen synthesis in hypertrophic scars. As these lesions regress after intralesional triamcinolone without suppression of synthesis, it is postulated that the drug acts by increasing the rate of collagen degradation. However, atrophy and depigmentation are annoying after intralesional triamcinolone injections, it usually reversible. The dermis will spontaneously thicken over a period of months.

Lehmann et al (1983) studied the corticosteroid atrophy in human skin with electron microscopy. They found that the primary effect of short-term steroid use was a rearrangement of the geometry of the dermal fibrous network. This was not due to alterations in the fibers themselves but a secondary consequence of the loss of ground substance. In the study, a 59% decrease in viable epidermal thickness was noted after the sixth week of treatment, as well as a flattening of the dermal-epidermal junction. The 3-dimensional architecture of the dermis was strikingly reorganized. This was largely brought about by resorption of the ground substance. Loss of ground substance resulted in decreased spaces between collagen and elastic fibers was shown by scanning and transmission electron microscopy. The fibrous network consequently collapsed, yielding a more compact papillary and reticular dermis. This compression caused the reorientation of both collagen and elastic fibers. However, no differences in collagen and elastin fine structure were noted. Fibroblasts were shrunken but not reduced in density. A marked decrease in number of mast cells was noted in 3-week specimens and virtually no mast cells were observed after 6 weeks.

This was also mentioned by Jarrett (1974) that the atrophy of scar tissue was not due entirely to the inhibition of collagen synthesis, but that there was also an increase in the breakdown of collagen. In his in vitro experimental culture of fibroblasts, the fibroblasts can be induced to synthesize collagenase by the action of corticosteroids. This steroid-induced collagenase appeared to be readily released into the surrounding dermis, and this would account for the rapid removal of the collagen. Furthermore, they showed that only the younger fibroblasts could be induced to produce collagenase: the older cells appeared to have lost the ability to respond.

Overdose of intralesional injection of steroid will lead to a feature of atrophy in which there is thinning of both epidermis and upper dermis. This preatrophy due to steroids has been equated with an early demonstration of a greatly visible subpapillary plexus (Ryan 1993).

Krusche & Worret (1995) studied the mechanical properties of 17 keloids in 9 patients before and during treatment with intralesional triamcinolone acetonide by using a commercially available noninvasive suction device for measuring skin elasticity in vivo. There was a marked decrease in the ratio between the viscous and the elastic deformation of the skin; and a less pronounced increase in the ability of the skin to return to its initial position after deformation being observed after three injections of triamcinolone acetonide. The findings indicate that the main effect of intralesional steroids on the connective tissue of keloids is a decrease in viscosity due to a loss of ground substance.

4.5 Pressure therapy

Ketchum & Cohen (1974) suggested the pressure applied should be maintained for a minimum of 4 to 6 months to reduce the incidence of recurrence.

Kischer et al (1978) indicated that the effects of pressure on numbers of mast cells may be identified in the papillary dermis, upper, and middle reticularis, since, under pressure these areas have similar mast cell counts as mature scars. However, the lower reticularis is not similar in mast cell counts to the corresponding area of mature scars. This suggests that the effects of pressure therapy are not realized at this depth until a much later time, perhaps the time at which constant pressure is no longer necessary, and final permanent resolution of the hypertrophic scar has taken place.

Although sulfated mucopolysaccharides have been found in mast cells it seems doubtful the amount would be enough to account for the 14-fold increase of chondroitin sulfate A in hypertrophic scars over that in normal skin. However, it should be significant that the levels of mast cells in hypertrophic scars are maintained throughout the period of hypertrophy, which often is years. This suggests a supportive or complementary role of the mast cell for hypertrophic scarring. Such an intimate relationship is further supported by the significant reduction of mast cells under pressure treatment, which mechanically resolves the hypertrophic scar.

Larson et al (1973) studied the effect of continuous pressure over the healed burned areas. Those areas with pressure compared to the areas without pressure revealed no nodular formation, loose collagen bundles, more interstitial space, less chondroitin sulfate A, and less cellular pattern.

Kischer et al (1975) studied the alteration of hypertrophic scars induced by mechanical pressure. It was suggested that hypertrophic scars and contractures might be rapidly resolved through application of pressure and forced extension. Examination of pressure-treated scars by scanning and transmission electron microscopy demonstrates a reduction in intercollagen cohesiveness and increasing numbers of vesicular fibroblasts. Assays of chondroitin sulfate show a decrease from the excessive levels found in untreated hypertrophic scars. It is suggested that the application of pressure increases an already present condition of hypoxia, which results in degeneration of many fibroblasts. The ratio of collagen synthesis to degradation would then be altered in favor of the latter, resulting in resolution of the scar.

Linares (1996) summarized the effect of pressure on hypertrophic scar. A granulation tissue showing collagen fibers in a disorganized, whorl-like arrangement will develop into hypertrophic scarring. If pressure is applied for a variable period of time, however, the collagen fibers will modify their disorganized orientation and will adopt a definite parallel arrangement which is characteristic of the pattern of the granulation tissue which leads to normotrophic healing. Likewise, under constant pressure, the whorl-like

arrangement of collagen fibers and nodular formation seen in immature hypertrophic scars assumes a progressive predominantly parallel arrangement similar to that found in normotrophic scars. Fig. 25 shows polarized light pictures of collagen fibers and the change of orientation due to pressure (Linares 1996).

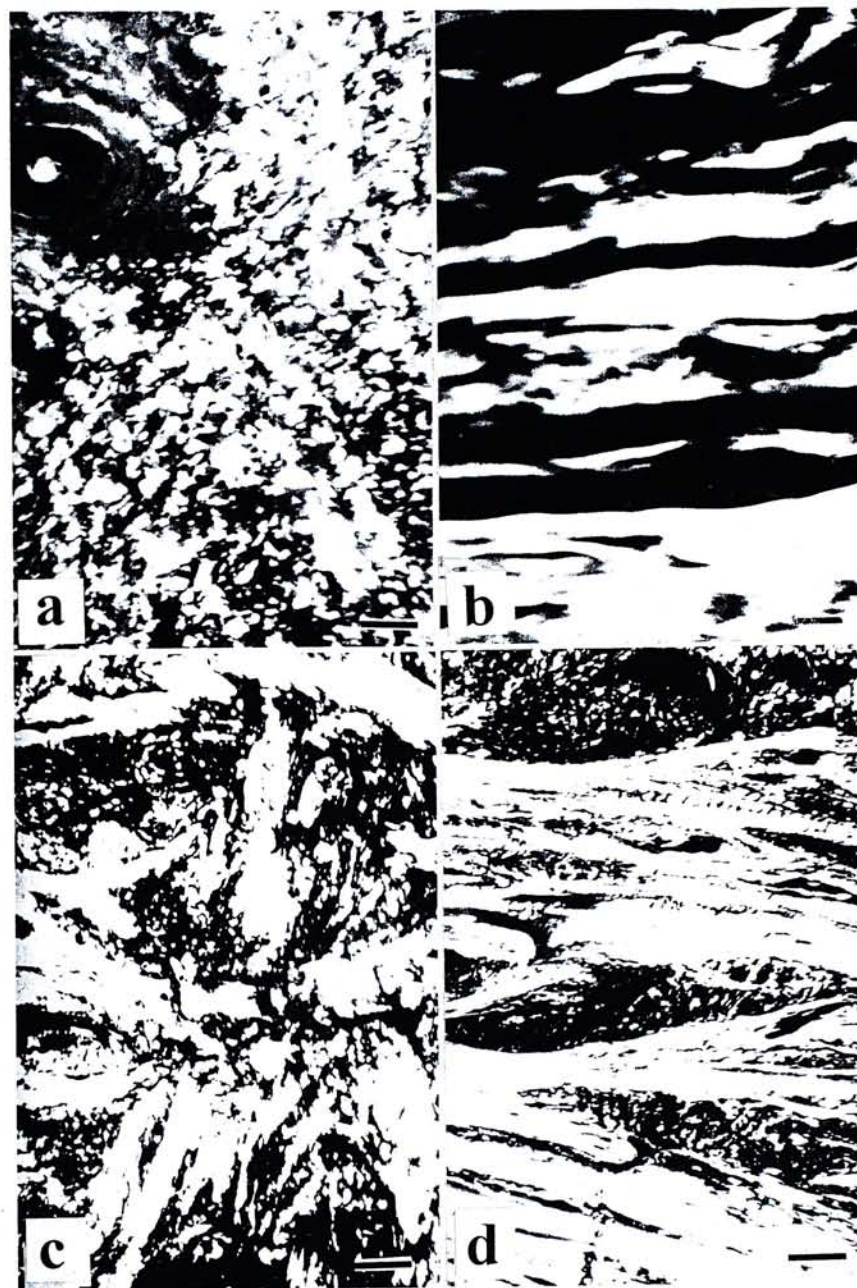


Fig. 25. Polarized light pictures of collagen fibers depicting
 (a) the tridimensional pattern of collagen in normal skin,
 (b) the parallel orientation in normotrophic scars,
 (c) the whorl-like arrangement in hypertrophic scars, and
 (d) the 'parallelization' of collagen fibers after pressure.
 After Linares 1996, *Total Burn Care*, p.394.

Constant pressure will decrease the vascularity, decrease the tissue partial pressure of oxygen, decrease the amount of proteoglycans, and decrease the cellular response as well as the collagen deposition.

Hambleton et al (1992) studied the effectiveness of pressure therapy in the treatment of hypertrophic scars by using ultrasound scanning technique throughout the initial healing, hypertrophy stages to maturation of the scars. The data confirmed that the flattening of scars observed after pressure therapy is indeed a decrease in the thickness of skin and not an embedding of fibrous tissue into underlying tissue to achieve a smoother contour. However, the thickness in the injured area was still greater than that observed in normal skin. The paling observed on maturation implies that a part of the flattening is a result of a reduction in the vascular content of the scar. The study of the mature scar indicated that the further reduction in scar depth is minimal. Pressure therapy is effective in hasten the scar maturation; many patients gain greater comfort and mobility from wearing pressure garments as soon as their burn wounds are healed. Itchiness of the healed areas may be relieved; patients with lower limb injuries reported improvement in walking and standing time and many adult patients and parents of burned children appreciated the protection the garments afforded whilst the newly healed skin was so vulnerable.

4.6 Topical silicone gel

McNee (1992) summarized some research done on the effect of silicone gel in the treatment of hypertrophic scars. The silicone gel softens, reduces and blanches scars. Quinn (1987) recommended the use of silicone gel for 24 hours/day in the treatment of hypertrophic scars but not for children with age under 5 as skin break down was noted doubled in such group of subjects.

Quinn et al (1985) investigated the four properties of silicone gel :

4.6.1 Mechanics

The silicone gel has an extensibility of $41.8 \pm 2\%$ ($n=4$), which is similar to skin (40%). It is sufficiently elastic to cover joints and allow movement.

4.6.2 Bacteriology

In the culture tests of organisms, silicone is demonstrated as impermeable to certain standard organisms (including pseudomonas, staph. aureus, escherichia coli, staphylococcus and streptococcus), and is inert since it neither inhibits microbial growth nor alter them in any way.

4.6.3 Water-vapor transmission rate

In the evaluation of the rate of water-vapor transmission through silicone gel, the result obtained indicate a water-vapor transmission rate of $4.5 \pm 1 \text{ g.m}^{-2} \text{ h}^{-1}$ ($n=10$), which is approximately half that of skin ($8.5 \pm 0.5 \text{ g.m}^{-2} \text{ h}^{-1}$).

4.6.4 Appearance in the Scanning Electronic Microscope

The surface of silicone gel is relatively flat with no pores under the SEM.

The mode of action of silicone gel in the treatment of hypertrophic scar is still under investigation. It has been shown not to involve pressure, temperature, oxygen tension or occlusion. However, studies by Quinn (1987) has demonstrated that a low molecular weight silicone fluid is leached from the Dow Corning silicone gel. By elimination, the mode of action of silicone gel must involved a chemical factor. It is believed that the silicone fluid hydrates the stratum corneum and alters the chemical structure of hypertrophic scars, thus reducing their thickness and color. It may also due the relatively impermeable silicone gel acting in the same way as stratum corneum and hence restores homeostasis to the burn scar, reducing capillary hyperemia and secondary fibrosis and hypertrophic scar formation.

Ahn et al (1989), Carney et al (1994) also reported the efficacy of using silicone gel. The use of silicone helps to reduce the vascular content of scar,

hasten the scar resolution, make it softer and hence a statistically significant improvement in the elasticity was reported. Ahn (1991) reported that there was an increased scar elasticity after receiving one and two months treatment in comparison with the controls. There was corresponding improvement clinically that persisted for at least 6 months.

4.7 Prevention of hypertrophic scar and scar contracture

Excision of the wound with successful autografting appears to permit the wound to heal without granulation (Abston 1987). There were experiences reported a decreased incidence of hypertrophic scars with early tangential excision. Other observation of burn wounds treated by tangential excision and covered with meshed skin grafts, the epithelium at a thickness of one to two cell layers. The pattern of the collagen in the underlying tissue developed parallel to the surface, and there were no compact nodules or whorls characteristic of older granulation tissue or hypertrophic scars.

When a hypertrophic scar occurs across a joint, contracture will limit the joint mobility. If the hypertrophic scar is left untreated, it will eventually undergo maturation, and the skin web will prevent motion. Eventually, shortening of the muscle and fibrous contracture of the joint capsule may make the contracture irreversible. When the size of the burn wound and the patient's condition permit, excision and grafting are within the first week and will permit the wound to heal without granulation.

In addition, hypertrophic scars and contractures can be prevented after grafting with attention to positioning, use of splints and elastic garments, and regular exercises. A well-planned exercise program in adjunct to pressure therapy is important throughout the hospitalization and is begun soon after admission to prevent contractures and maintain mobility.

Cheng et al (1984) studied the usefulness and fallacies of pressure therapy. Several recommendations had been made to improve the techniques, including the standardization of measurement techniques and garment tailoring, a regular checking of pressure at the garment-scar interface using pressure transducers, an appropriate garment adjustments, a strict regimen for garment wearing, and the intelligent use of pressure-padding and reinforcement.

Ahn et al (1991) conducted a cohort to study the change in scar volume in 21 surgical incisions in response to silicone gel treatment. The gel-treated incisions gained less volume than control incisions. Clinical assessment confirmed this quantitative demonstration of a decrement in scar volume. Therefore, topical silicone gel is also effective in the prevention of hypertrophic scar.

In short, surgery, corticoids, and pressure, alone or in combination, are currently the most universally used methods of treatment.

5 *Assessment tools for Hypertrophic Scar and the Clinical Application*

The post-burn hypertrophic scar are rigid and inextensible, during the maturation process, the thickness will decrease and become softer gradually.

Subjective clinical scores have been used to assess the change of mechanical properties and thickness of scar (Leung et al, 1984; Sullivan et al, 1990).

Pathological changes were assigned scores on an index scale base on their deviation from normal (rated 0). The Vancouver Scar Scale (modified from the Vancouver General Hospital Burn Assessment Scale) is widely used in clinical practice and research document change in scar appearance. Assessment items consisted of comparing the vascularization, pliability, scar height and even pigmentation.

In order to monitor the efficacy of different treatment modalities, objective and quantifiable assessment tools are essential.

5.1 *Clinical Observation of the appearance*

Hypertrophic scars are thick, rigid, red scars that become apparent six to eight weeks after spontaneous healing. After one or two years, hypertrophic scars soften, flatten, and lose the hyperemia (Abston 1987). During clinical observation, the change in skin colour, scar height texture is considered.

Hypertrophic scars are thickened, tumourous, hard areas of scarified skin which are notoriously rigid and inextensible. Scar maturation is associated with the clinical evidence of progressive softening and thinning of the scar. Therefore,

by measuring the mechanical properties of such tissue, monitoring of scar maturation was thought to be possible.

5.2 Ultrasonography and thickness

5.2.1 Ultrasound

Ultrasound is sound with frequencies above audible range, reflected and scattered back from tissue interfaces (Griffiths & Short, 1994). The images of subcutaneous tissue is produced electronically on a video-screen.

Goldstein (1990) defined ultrasound waves are mechanical pressure waves similar to audible sound waves and must have a medium in which to propagate. The frequency (f) is the number of high- or low-pressure regions crossing each area of tissue each second. The frequencies of the sound waves used in medical ultrasound are much higher than the human audible range (20 to 16,000 hertz); hence, the waves are called ultrasound.

The acoustic velocity (c) of an ultrasound wave is the wave velocity of the pressure waves traveling through the propagation medium. Acoustic velocity is essentially frequency independent with an average value of 1540 meters per second. Most soft tissues in the body have acoustic velocities within 3% of this average.

Longitudinal acoustic waves are waves in which the direction of particle motion is parallel or anti-parallel to the wave velocity. Transverse waves are waves in which the direction of particle motion is perpendicular to the wave velocity. Longitudinal acoustic waves are the only waves that will propagate in a fluid (liquid or gas), so only longitudinal acoustic waves can propagate in the soft-tissue structures of the body.

The acoustic wavelength is the basic repetition distance in space for a single frequency wave, joining points of equal phase. In ultrasound the term phase refers to the time an event occurs, such as the exact point in the acoustic cycle when a certain pressure is attained. Due to their definitions, the wavelength and frequency of acoustic waves are related to each other by the following standard equation.

$$c = \lambda f$$

$$\text{Acoustic velocity} = \frac{\text{acoustic wavelength}}{\text{frequency}}$$

The acoustic pressure amplitude at a point in space (P) is the particle pressure, which is the difference between the pressure when the wave is present and the ambient pressure. The acoustic intensity (I) is defined as the energy (power) propagating through a unit area in the medium per unit time.

The acoustic impedance (Z) of the propagation medium is important in predicting the magnitudes of the reflected echoes at interfaces between two different tissues.

$$Z = \rho c$$

$$\begin{array}{lcl} \text{Acoustic} & = & \text{tissue density} \quad \times \quad \text{Acoustic velocity} \\ \text{impedance} & & \text{g/cm}^3 \quad \quad \quad \text{cm/sec} \end{array}$$

Because both tissue density and acoustic velocity are independent of frequency, acoustic impedance is also frequency independent and relies only on the tissue's mechanical properties.

5.2.2 Pulse-echo distance measurement

The slowness of acoustic velocity in tissue permits distance measurements to be made in a novel manner. The time it takes for a pulsed sound wave to travel from a transducer to a reflector and back can be measured and then, along with the known acoustic velocity, can be used to calculate their separation :

$$R = \frac{1}{2} ct$$

$$\text{Distance} = \frac{1}{2} \text{ Acoustic velocity} \times \text{time of flight of sound wave}$$

The factor of one half in the equation comes from the fact that the sound waves have actually traversed the range twice, once as transmitted sound and once as reflected sound.

5.2.3 Echo generation

Ultrasound echoes are produced by two types of reflectors : specular and diffuse. High-amplitude echoes are usually due to specular reflectors; low-amplitude echoes are due to diffuse reflectors.

Diffuse reflectors have physical dimensions that are much smaller than the acoustic wavelength. Point reflectors and interface roughness are common examples of diffuse reflectors. Diffuse reflectors scatter ultrasound in all directions, producing low-amplitude echoes. Slight local variations in acoustic impedance inside body organs act as diffuse reflectors. Modern ultrasonic scanners use a gray scale presentation in which image shades of gray represent the received range of echo amplitudes.

Specular reflectors are large mirrorlike interfaces in the body between two different soft tissues. If a sound ray (direction of travel of the short sound pulse) is incident on an interface, two rays are produced: a transmitted ray that propagates into the second soft tissue and a reflected ray that propagates back into the first soft tissue.

The direction of the reflected ray (echo) is governed by the law of reflection that states that the angle of incidence equals the angle of reflection. For single transducer pulse-echo equipment to register the reflected echo, it must be backscattered at an angle of 180 degrees and

back to the front face of the transducer. So only when the incident beam is perpendicular to the interface will the reflected beam be received by the transducer (the angles of incidence and reflection are both 0).

The reflection coefficient is defined as the fraction of sound intensity reflected from the reflector (target tissue). At normal incidence on a specularly reflecting interface, the reflection coefficient is given by the expression

$$R = (Z_2 - Z_1)^2 / (Z_2 + Z_1)^2$$

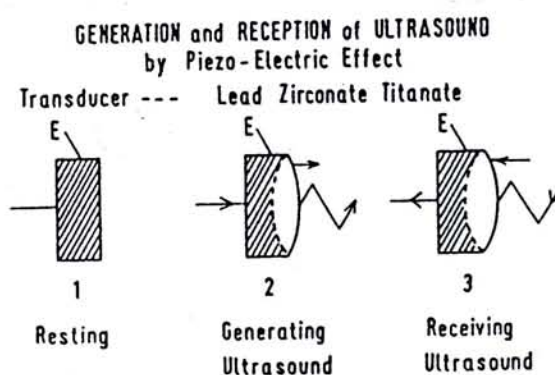
where the wave is propagating in medium 1 and meets a specular interface with medium 2. The strength of the reflection coefficient is governed uniquely by the acoustic impedances of the two different tissues at the interface. The strength of the reflection coefficient really depends on the ratio of the acoustic impedances at the boundary, and not on their absolute magnitude. The reflection coefficient is of the same magnitude for both the ratio of the acoustic impedance and its reciprocal. The reflection coefficient is zero (no reflection) if the ratio of acoustic impedances at the boundary is unity. This is known as impedance matching and maximum energy transmission results with no reflection. The larger the difference in acoustic impedances at the boundary, the larger the magnitude of the reflection coefficient. This is known as impedance mis-matching and the more the acoustic impedance mis-match, the larger the reflection coefficient.

The acoustic impedance is related to the density of the tissue hold. Figures shown that fat has a lower mass density than soft tissue, and bone has a much higher mass density than soft tissue. The acoustic impedance of the tissue follow the same pattern correspondingly.

5.2.4 Transducer beam pattern

A diagrammatic description of the mechanism was suggested by Sutton (1994). The ultrasonic waves are produced from a transducer and travel through human tissues at a velocity of some 1500 meters per second. When the wave reaches an object or surface with a different texture, or acoustic nature, a wave is reflected back. These echoes are received by the apparatus and changed into electric current, amplified and shown on a cathode-ray tube. Fig. 26 shows the generation and reception of ultrasound. (Sutton 1994, Radiology & Imaging for Medical Students p.13)

Fig. 26



Generation and reception of ultrasound. 1. The transducer (hatched) coated with conducting material is in a resting phase. 2. A voltage (\rightarrow) is applied to the transducer surface. The transducer resonates in response and it produces ultrasound from its surface (\wedge) (direct piezo-electric effect). 3. A pulse of ultrasound (\wedge) strikes the surface of the transducer which resonates as a result. A voltage (\leftarrow) is generated on the transducer surface (converse piezo-electric effect).

When the transducer is moved over the skin the series of linear dots are frozen as bright lines which form a two-dimensional image representing a linear section of the organ under examination. Nowadays, many commercially available ultrasound machine are real time two-dimensional scanning, which make use of an automatic scanning mechanism to produce the images in "real time", i.e. actual motion is shown as it occurs.

5.2.5 Ultrasound instrumentation

Most commercially available ultrasonal imaging equipment are grey scale using time delay and echo amplitude information in creating the image. The basic-building block circuits contains transmitter, receiver, display and scan converter. Fig. 27 shows the ultrasonic equipment; circuit building blocks (Goldstein 1990).

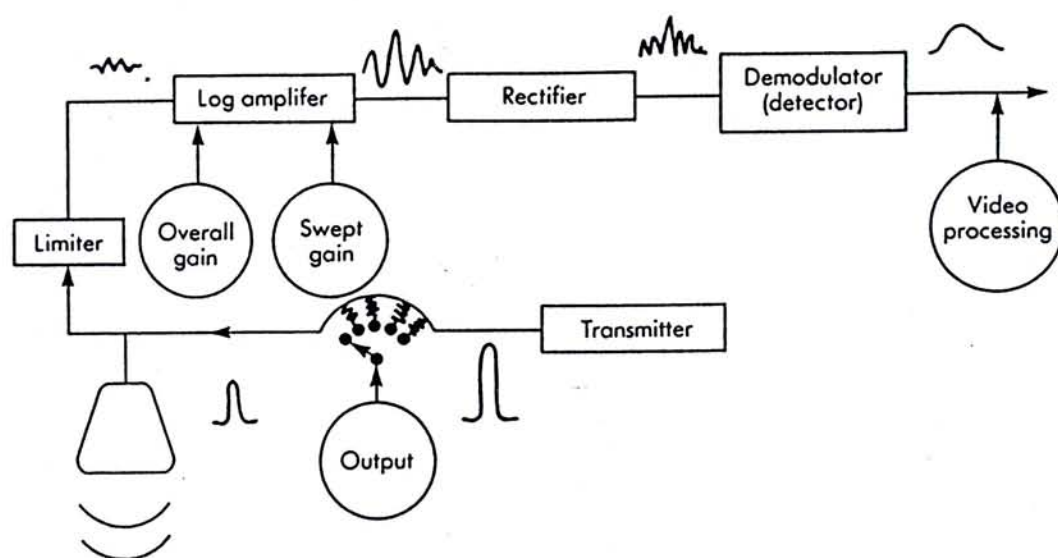


Fig. 27 Ultrasonic equipment; circuit building blocks. The transducer is shown transmitting into the patient. The circles indicate operator controls that affect the echo amplitude data written into the ultrasonic image. At the top of the figure, the echo waveforms are presented at various stages of signal processing.

The transmitter circuit produces the pulses. The receiver will receive the echoes and transmission occurs in the transducer. The build-in limiter and log amplifier will protect the receiver by high-amplitude signal that is above a certain threshold, and amplifies the weak signals respectively. Therefore, both the low- and high- amplitude echoes may be seen in the same image. Then, the echo signals enter the rectifier to control the output so that the positive and negative half-cycles will not be cancelled out, and then the demodulator to smoothen the circuit. The ultrasonic images are displayed through Cathode ray tubes (CRT), or broadcast TV tubes in form of a two-dimensional format, the echo data will be displayed as brightness.

There are several ultrasonic display mode. The A-mode (amplitude mode), displays give a graphic presentation of echo amplitude information. The B-mode (brightness) display the echo data on the two-dimensional CRT face. The brightness spot represents the amplitude of the received echoes and give a gray scale image. The M-mode (motion) is a B-mode display in motion, the vertical B-mode display line represented the distance whereas the brightness represented the amplitudes.

On the market, the analog scan converters and digital scan converters are available. While the latter is proved to be relatively inexpensive, reliable, and flexible in data handling capabilities. The "real-time"

scanning allow the observation of a dynamic moving image on the screen , and hence provide an immediate feedback of the image (Squire (1982).

5.2.6 Application of ultrasound in the study of hypertrophic scar thickness

Alexander & Miller (1979) had presented the use of a high frequency, high resolution ultrasonic echo technique in determining the thickness of human skin. The "ultrasonic biometric ruler" is shown to provide an accurate, simple, noninvasive method for measuring full-thickness human skin. In addition to the determination of skin thickness, it is demonstrated that the underlying subcutaneous fat and muscle can also be non-invasively "explored" with the possibility of identifying a variety of skin and underlying tissue lesions by observing the attenuation of signal and the emission of minor echoes. Alexander & Miller compared the ultrasound method with a proven radiological method. A correlation coefficient of $r=0.99$ was computed. A rank correlation coefficient of $r_s=0.91$ was obtained making the correlation significant at the 95% confidence level.

The advantages of using the ultrasound techniques include :

- it involves a direct visualization of a small segment of unloaded skin,
- being usable any place on the body regardless of cross-sectional depth and skin-fold availability,
- not involving any radiation hazard, and
- low cost per test when compared with the radiological technique.

It is suggested that ultrasound device is completely non-invasive, allows a comparison of the thickness of dermal tissue in the traumatized area with that in the normal skin at regular intervals. The readily distinguishable pulsed ultrasound echoes can be obtained from skin, subcutaneous fat and muscle and hence making the thickness of the skin can be accurately measured with this method.

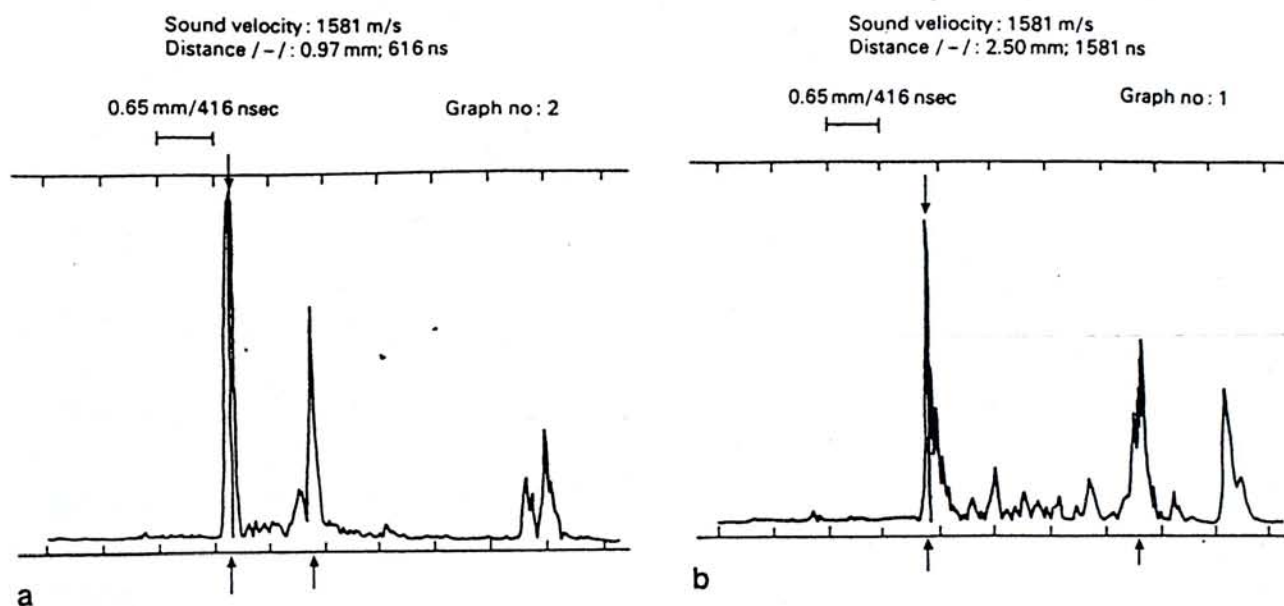
Ultrasound has been utilized both clinically and experimentally by Søndergaard, et al (1985), to assess other dermatological conditions such as psoriasis and malignant melanoma.

High frequency ultrasound scanning was used (Dermascan A equipment) by Hambleton J. et al, (1992), to measure the thickness of hypertrophic scars following burn injury. Echoes were relayed to a visual display unit in form of electrical impulses.

The distance between the peaks correlates with the skin thickness (depth) in millimeters. (Fig. 28)

Fig. 28 Ultrasound images from normal skin (a) and hypertrophic scar (b).

The distance between the arrows correlates with the skin thickness (depth) in millimeters. This value is shown at the top of the screen below that for the sound velocity.



This study confirmed that the raised hypertrophic scar is related to an increased in the thickness of scar tissue following burn injury compared with normal skin.

Sawada (1994) has developed a technique to measure the height and size of the scar by making cast models. As the scar tissue may extend to the invisible subcutaneous layer, this method can only compare the general appearance of the scar clinically.

This is further supported by Fong et al (1996), 99 scars of 35 patients were studied. The ultrasonically measured and the clinically estimated height of scars were compared. The mean score of clinical estimation of scar height was significantly smaller than that of the ultrasonal measurement. However, a good, direct correlation was shown between ultrasonal measurement and clinical estimation when the respective scar height grading was added to the overall clinical rating. In addition, there was a tendency for clinical under-estimation of scar height, as the scar usually extends underneath the invisible layer.

5.3 Elastometry (Cutometer) and elasticity

The mechanical properties of the skin reflected the behaviour of the basal elements, organisation of the elastic collagen networks. Methods that permit measurement of the mechanical parameters of extensibility and elastic recovery included :

- deformation applied perpendicular to the plane of the skin (indentation, levarometry, ballistometry, and suction)
- constraint applied parallel to the skin surface (uniaxial extensibility, torsion, and elongation vibration).

The latter methods allow interpretation of results independent from the attachments and the influence of the subcutaneous tissues (Rigal & Lèvêque 1993).

Cutometer is a suction device evaluates the mechanical properties by recording the strain applied skin surface. An adjustable pressure ranged from 20 to 500 mbar can be provided through the measurement probe. The skin is then being drawn into the opening of the measurement probe, with standard diameter of 2 mm. The two build-in optical lenses positioned at the opening of the measuring probe will measure the depth of the penetration skin without any friction mechanical effect. The strain-time mode is characterized by an adjustable period of application of a certain vacuum, followed by a relaxation period. A graphical presentation of the skin extension as a function of time can be obtained (Ennen et al).

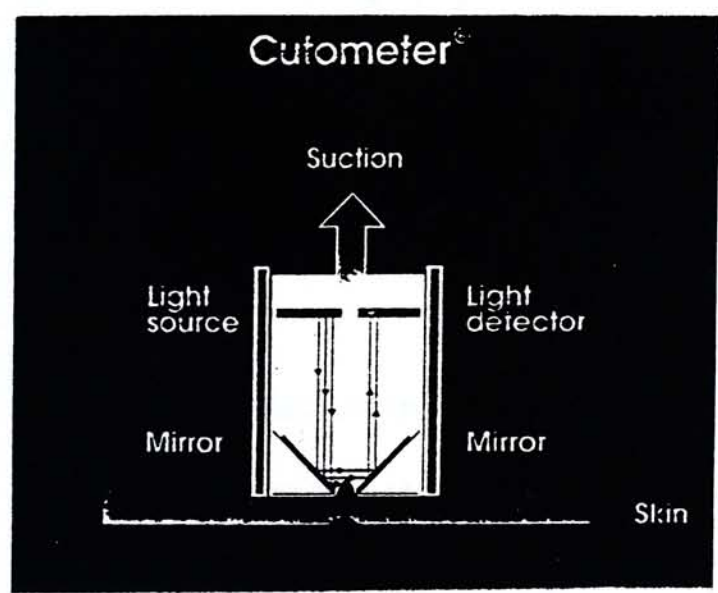
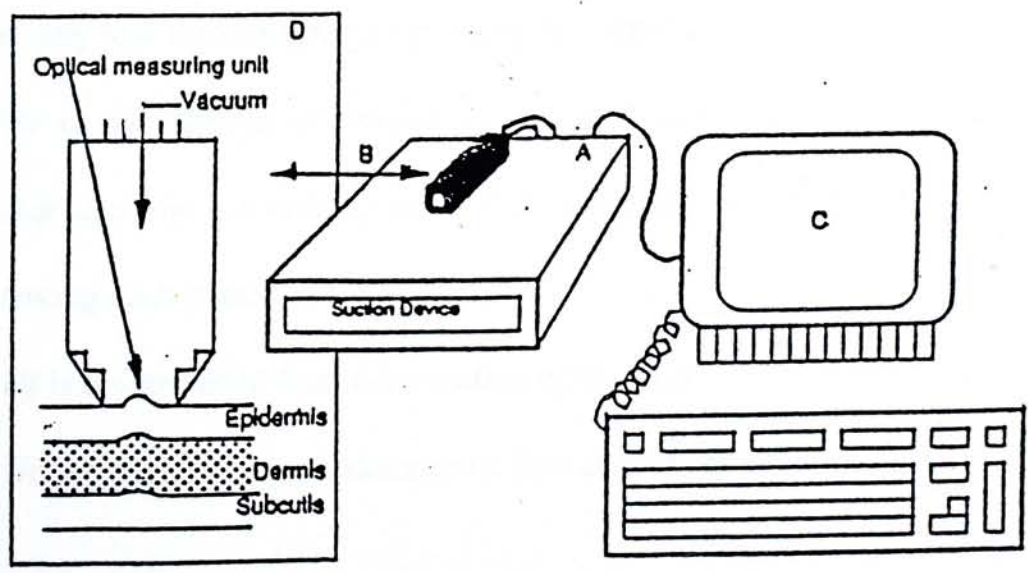


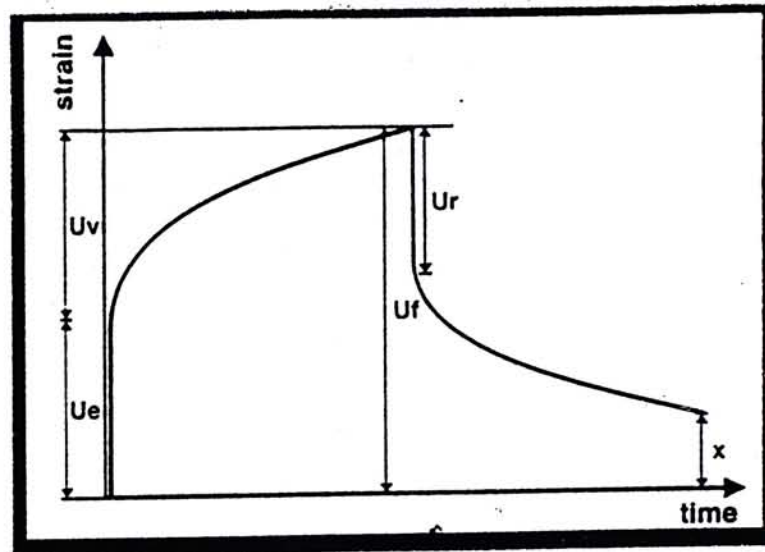
Fig.29
illustrates the suction device in the measurement of the elasticity :
a. the suction device
b. the measurement probe
c. the CPU
d. cross-section of the optical measuring unit



Elasticity is the property of a solid to deform due to an application of the load, and to regain its original dimension upon removal of the load. Viscosity describes the property of a fluid to incorporate a tension depending on deformation and velocity.

Fig.30

The strain-time curve



U_e is the immediate extension of skin due to the application of an external force at the early phase. Only the elastic component of deformation is obvious. The skin deformation is straightening and stretching of all fibres occurs. there is only low restoring force opposing the applied load.

U_v is the delayed distension of the final deformation phase. The viscous characteristic is a consequence of the inner friction of certain skin components among each other.

U_f is the extent of final deformation of the skin.

U_r represents the immediate retraction after termination of load.

Ennen et al has studied the effect of cutaneous aging and solar exposure on the change of skin extensibility. Two methods of equipments were used for comparison. There were suction and torsion devices. Similar graphic presentation were obtained in both methods. U_f (the elongation of skin due to pressure), U_v/U_e (ratio of viscous and elastic part) were analysed. However, direct comparison of the data was impossible as there is a significant difference in the diameter of the measuring probe (2mm in the suction device, 22mm in the torsion device). The volume involved in the torsional evaluation is 100 times the volume of the suction counterpart as it was suggested that a higher internal friction is involved in the torsion measurement.

Krusche & Worret (1995) also adopted this method to study the response of keloid to intralesional injection therapy. The parameters being observed including U_e , U_v , U_r & U_f (were skin thickness dependent values); and U_v/U_e , U_r/U_f ratios (parameters independent of skin thickness). U_v/U_e was view as the ratio between the viscous and the elastic deformation of the skin, was higher in comparison with normal skin. U_r/U_f representing the biological elasticity, the ability of the skin to return to its initial position after deformation is lower than the normal skin.

5.4 Application of elastometry

Longacre et al (1961) reported that in 85 diffractograms prepared in the Biophysical Laboratory of the Christ Hospital Institute of Research, there are two systems of collagen fibrils in the dermis, which represent a well-organized

biologic system, one running parallel to the wrinkle lines and the other perpendicular. The strongest orientation is derived from the collagen fibers parallel to the crease lines. Sections of skin taken from various parts of the body showed preferential orientation of the collagen in the same direction as the crease lines. Diffractograms obtained from mature linear scars showed orientation of collagen principally in the longitudinal direction of the scar. Diffractograms taken of the longitudinal sections of hypertrophic scars showed that the collagen was oriented in the longitudinal extension of the scar. In growing connective tissue and in scar there is the formation of new fibrils and the addition of more fibrils act as templates to extend the polymerization of collagen. Cross linkage with older fibers increases the strength. Eventually the main process will be the increase in the diameter of the fibers by the accretion of more tropocollagen molecules. The outer layers will be more loosely aggregated and hence more easily extractable.

Clark et al (1987) studied the extensibility and stiffness of hypertrophic scars. It was concluded that the clinical grading of the scar correlate well with the measurement. That is the worse (harder, more vascularized, thicker) the scar is, higher the value of Modulus of Elasticity and lower the value of extensibility. The hypertrophic scar was stiffer and less extensible. Along the natural remodelling process of hypertrophic scars, the scars will become more extensible.

Katz et al (1985) quantified the progress of hypertrophic burn scar. Tonometry and ultrasonography were used in the measurement.

In the study of the effectiveness of triamcnenolone acetonide on keloid, Krusche & Worret (1995) used the suction device to monitor the progress of the keloid responses. The viscous component in keloid tissue is found to be increased while the elasticity is decreased in comparison to normal skin. After injection of tiramcinolone acetonide, there is a reduction in the ratio U_v/U_e suggesting a decrease in the viscosity due to the effect of triamcinolone acetonide on the connective tissue. However, the change in the U_r/U_f ratio is not so dramatic indicated that there is not much improvemnet in the biological elasticity of the keloid.

Matsuzaki et al (1995) also adopted the same suction device to document the change of skin elasticity of the cultured epithelial autografting on meshed skin graft scars. Contralateral healthy skin of each studied site was selected as control. Only the value U_e , the elastic deformation was analysed in 4 cases. The results showed improvement in the appearance and the elasticity after resurfacing of the meshed skin graft with the cultured epithelium.

Chapter Four : OBJECTIVES AND METHODOLOGY OF THE STUDY

1 OBJECTIVES OF THE STUDY

- 1.1 To study the reliability of the ultrasonography and skin elastometry in the assessment of post-burn hypertrophic scar.
- 1.2 To find out the correlation of the results with traditional clinical grading.
- 1.3 To study the predictive value of the ultrasonography and elastometry through longitudinal measurements.

2 STUDY SUBJECTS

From December 1994 to June 1995, 18 consecutive burn cases were randomly selected into the present longitudinal study. 7 were adults and 11 were children with age ranged from 1 to 60. Among the 18 patients, 14 of them suffered from scald injury, others from flame burn, melted wax, cold and frictional burns respectively. 10 of them have received debridement, SSG or xenograft, and 8 of them were treated conservatively.

All of the cases were follow up monthly in the Occupational Therapy Department, Prince of Wales Hospital for pressure therapy. Patients will be given local tailor-made pressure garment to the corresponding area. Therapists will follow a

standard procedure to take the measurement of the body size, draft the pattern so as to provide circumferential pressure according to the equation.

Material being used for fabrication of the pressure garment is lycranet which is elastic in nature. The garment will be sewn with standardized method. Although garments for different region will differ in the pattern drafting and sewing procedure, the principle is to provide a therapeutic pressure of 20 - 25 mmHg. This pressure can be assured by checking with a pneumatic pressure sensor. After fitting the pressure garment, they were required to put on 24 hours a day and only taken off for hygiene measure. Since the elasticity of the garment material will be reduced with wear and tear, three sets of identical garments will be issued for rotational use. Therefore, the life of the material can be improved. The patients were also required to bring back the pressure garments to adjust the pressure every month, so that the pressure is kept as constant as possible. For some of the concave area, in accordance with the anatomical structure where it may not be able to exert the desirable pressure, a pressure localizer such as plastazote padding will be given (Leung & Ng 1980, Cheng & Evans et al 1983).

All cases were also actively attending the Pressure Garment Clinic which was jointly run by the Occupational Therapy Department, and the Department of Orthopaedic & Traumatology, Prince of Wales Hospital to monitor the progress of the patients. The doctors will assess the cases and arrange other additional treatment such as injection of cortico-steriod, surgical release if the scar does not respond to pressure

therapy. Since the burn patterns were so unique to every patient, a great variety of sites were involved. The body was distributed into 20 areas and coded (table 2).

Table 2

code no.	site of injury	code no.	site of injury
1.	ankle	11.	hand
2.	leg/calf	12.	groin
3.	knee	13.	buttock
4.	popliteal fossa	14.	back
5.	thigh	15.	abdomen
6.	hip	16.	chest
7.	arm	17.	neck
8.	forearm	18.	chin
9.	cubital fossa	19.	mouth corner
10.	wrist	20.	forehead

3 METHODOLOGY

According to Quinn (1987), the water vapour transpiration rate (WVTR) of skin is $8.5 \pm 0.5 \text{ g/m}^2/\text{h}$. This is affected by the change of the temperature and humidity. It is believed that the WVTR of scar will in turn affect the elasticity being measured. The Electronic Engineer of the Prince of Wales Hospital guaranteed that the whole building was under the control of a central air-condition plant, the temperature and relative humidity of each room were maintained within the range of 22 - 24 °C and 65 -75 % respectively. In order to minimize the environmental variations, measurements were performed in the same assessment room with controlled temperature (22 - 24 °C) and relative humidity (65 - 75 %), after the subjects had been taken off the pressure garment and physically inactive for at least 20 minutes.

Every scar was outlined with a transparent paper in the first appointment. The thickest site, usually in the middle of the scar (Linares 1996) where wound tension was

highest and took longer period of time to heal, would be marked. In order to ensure the consistency, the scar were marked according to the map in every follow-up session. Every individual will be reassessed monthly. Photographs, ultrasonographs and elasticity of the hypertrophic scar were recorded every time.

The elasticity of skin depends on skin tension and vary with different body regions. Stark et al (1977) stated that the skin tension depends on 3 factors: the nature of elastic fibres in dermis, the movements of the body and variations in bulk of the tissue covered. Hence, the scars on different body parts vary in surface contour, the position of the scar, movement/ contraction of muscle and the direction of measurement probe would also contribute to the variation of the result. In view of these, patients were required to rest the corresponding body parts on table or chair with the hypertrophic scar facing upwards. Then the measurement probes were placed perpendicular to the scar. No extra pressure were exerted apart from the probe's own weight.

In order to ensure that patients could rest the body parts with the hypertrophic scar facing upward, the patients were recommended to rest as shown in Table 3 :

Table 3

hypertrophic scar on different body parts	positioning
• dorsum of foot	patient sit on plinth, & rest the foot on chair
• ankle	side lying/ prone lying
• leg	supine/ prone lying
• knee	long sit on plinth
• thigh	supine/ prone lying
• trunk	supine/ prone lying
• elbow/arm/forearm	rest the upper limb on table with the scar facing upward
• hand	rest the hand and forearm on table with the scar facing upward

In order to minimize the inter-observer error, all measurement were taken by a trained therapist throughout the study, under the guidance of an experienced radiologist.

Measurement include ultrasonography (for thickness), cutometer (visco-elasticity) and clinical rating (change in colour, consistency and other symptoms like itchiness, hypersensitive, and blistering).

The calibration of the machines were important to ensure the reliability of the tools. The ultrasound machine was calibrated by the manufacturer in accordance with the international standard, RMI, phantom model 413A. High reliability was guranteed by the manufacturer. For the cutometer, it was started at least 30 minutes before each session and calibrated with standard procedures according to the manual. Hence, the pressure was kept at 500 mbar for each measurement.

4 ASSESSMENT OF THICKNESS OF HYPERTROPHIC SCAR

The thickness of scar were measured by ultrasonography (Hambleton et al 1992, Katz et al 1985, Sawada 1994). When the ultrasound waves pass from one tissue category to another, a portion of the sound wave is reflected. The reflected sound wave is detected by a transducer and transformed to an electrical impulse and becomes the image appearing through a computer interface. The time taken for these reflections to return to the transducer is a function of the distance to one tissue interface and another (Sung et al 1992, Goldstein 1988, Rumack et al 1994). Since the density and structure of hypertrophic scar tissue is different to its tissue adjacent and underneath, the thickness and the boundary of the hypertrophic scar could easily be revealed by the reflected waves. In the study, a portable ultrasound machine (Aloka SSD 500) with electronic linear probe (UST-5512U-7.5MHz/38mm) is adopted. The measurement give an accuracy up to 0.1cm. The directions of the measurement probe will affect the image display. All the ultrasound images taken are transverse view of the hypertrophic scar with reference to the anatomical position.

Fig. 31, Diagram to show cross-section of normal skin.

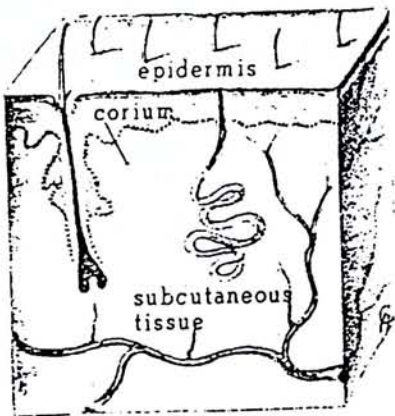


Fig. 32, Diagram to show ultrasonograph of normal skin

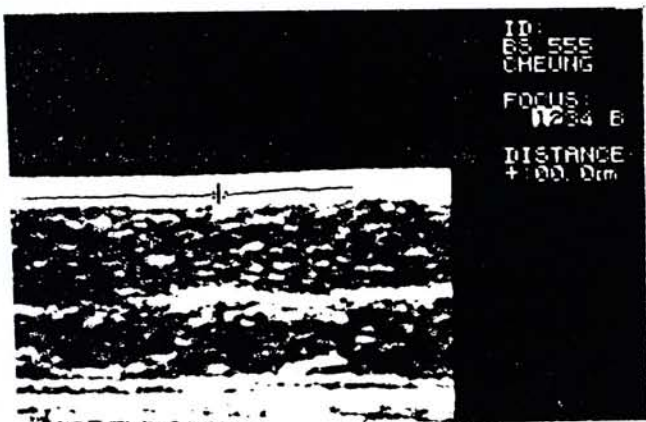


Fig. 34, Diagram to show ultrasonograph of scar tissue

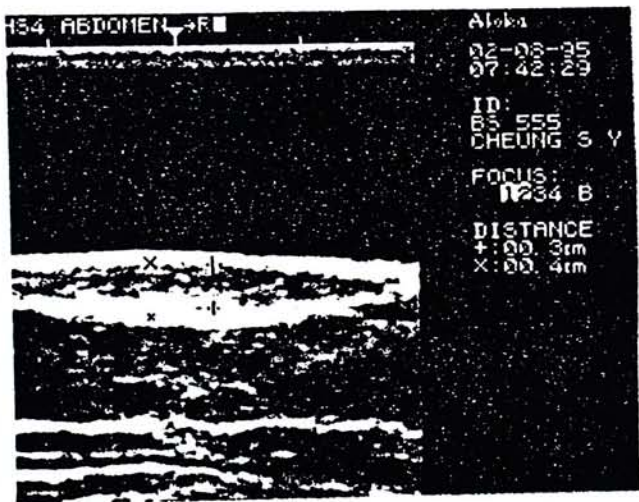


Fig. 33, Sketch of normal skin according to the ultrasonograph

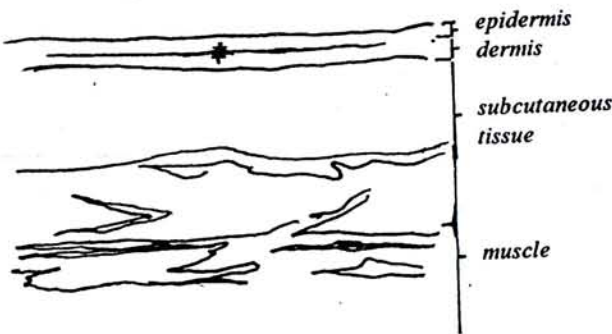
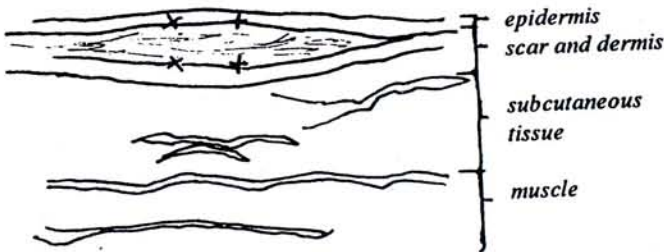


Fig. 35, Sketch of scar tissue according to the ultrasonograph

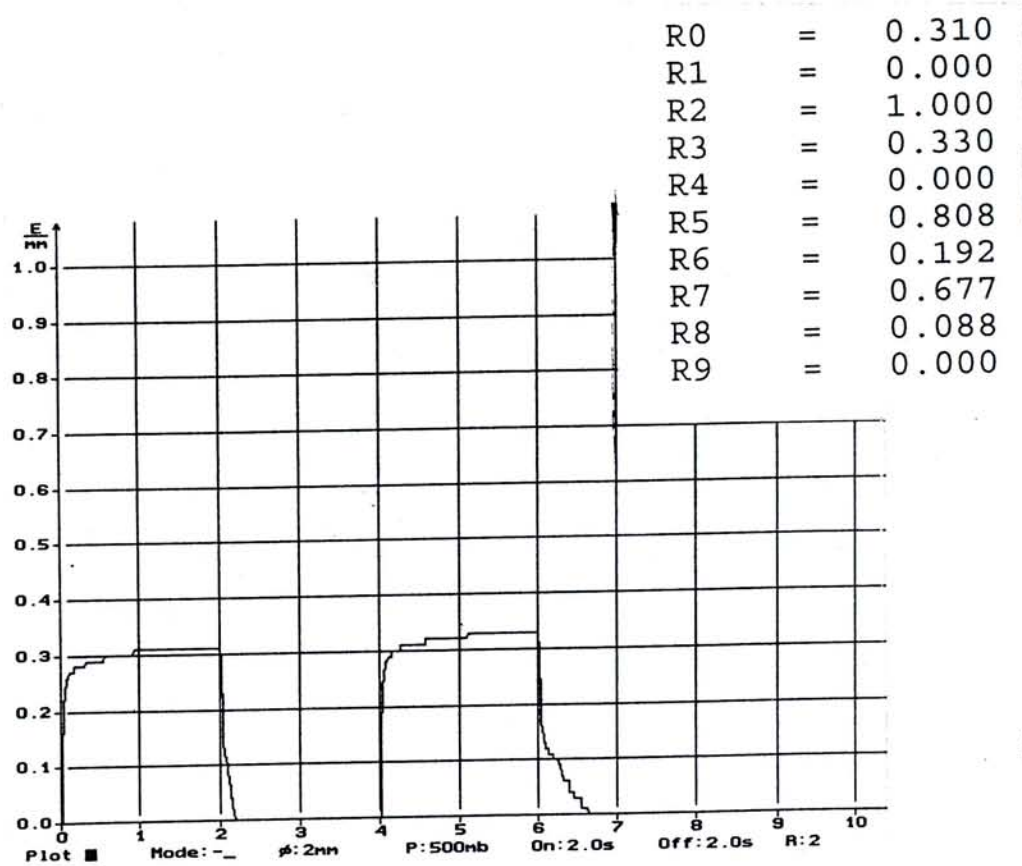


The thickest part of the scar tissue will be marked with the ultrasonic callipers and the thickness of scar tissue can be computed automatically.

5 **ASSESSMENT OF VISCO-ELASTICITY OF HYPERTROPHIC SCAR**

Cutometer (SEM 575, Courage and Khazaka, Cologne, F.R.G.) is also used as it is a non-invasive, in-vivo suction device. It is connected to a computer for control and display of the measurement. The instrument measures the vertical deformation of the skin surface when the skin is pulled in the circular aperture (2mm in diameter) of the measuring probe after application of a constant suction pressure (500 mbar) for 2 seconds. Then the negative pressure is cut-off and the skin can return to its original shape. There is a built-in optical system in the measuring probe which generate and receive the reflected infrared light beam. The depth of skin penetration (value R0) is measured through the diminution of light intensity in function of skin penetration (Elsner et al, 1990). Then a strain-time curve (Fig. 36) could be obtained with the deformation of the skin (strain) display as a function of time.

Fig. 36

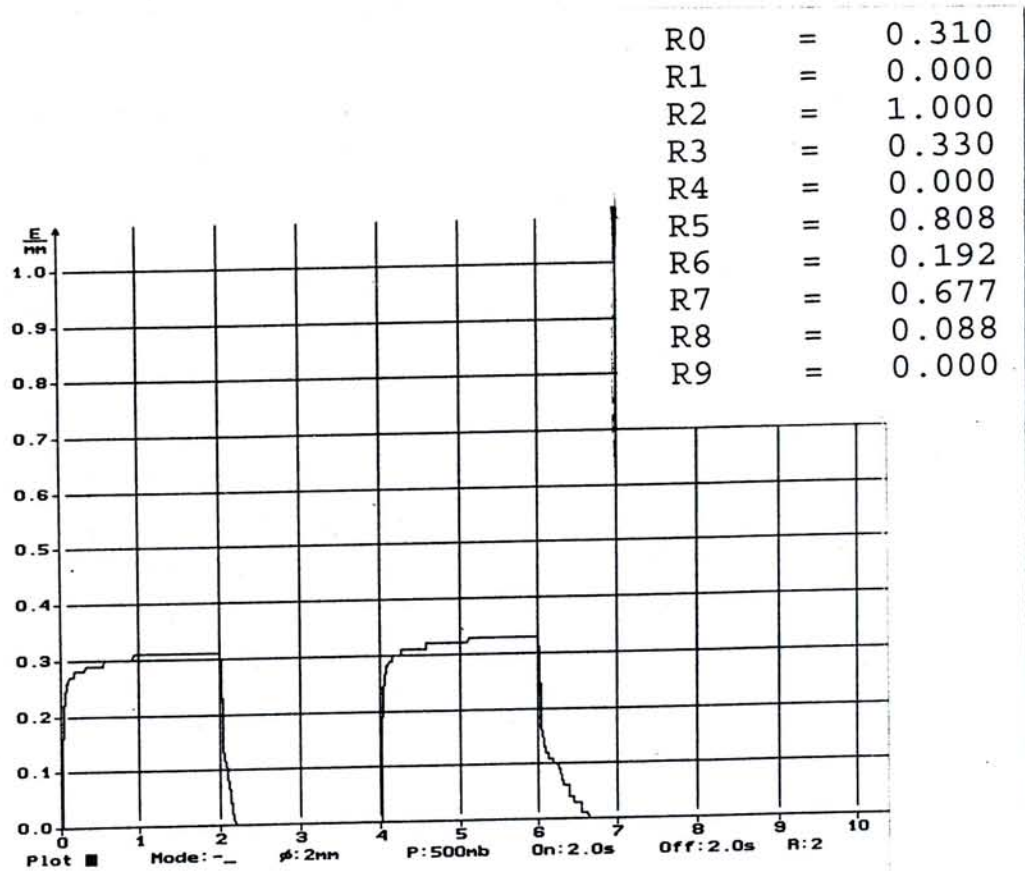


A build-in database file (CT.dbf) can read and calculate R0, R2, R5, R6 and R8 automatically.

5 **ASSESSMENT OF VISCO-ELASTICITY OF HYPERTROPHIC SCAR**

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Fig. 36



A build-in database file (CT.dbf) can read and calculate R0, R2, R5, R6 and R8 automatically.

In the assessment of normal skin, Cua et al (1990) suggested that the immediate distension (U_e), delayed distension (U_v), immediate retraction (U_r) and final distension (U_f) are a function of skin thickness. Hence different regions of a body will have different result. However, some biologically relevant ratios can be regarded as skin thickness independent.

The meaning of the reading being considered in data analysis :

$R_0 = U_f$, the total deviation of the skin

The skin deformation (R_0) can be measured by this optical system up to an accuracy of 0.10mm

$R_2 = U_r/U_f$, the gross elasticity, the ratio between immediate retraction and total distension representing the skin's ability to recover to its initial position after deformation, and

$R_5 = U_r/U_e$, the net elasticity, the ratio of the immediate retraction to the immediate distension. This ratio is considered as a biological important factor for the characterization of elasticity of the skin.

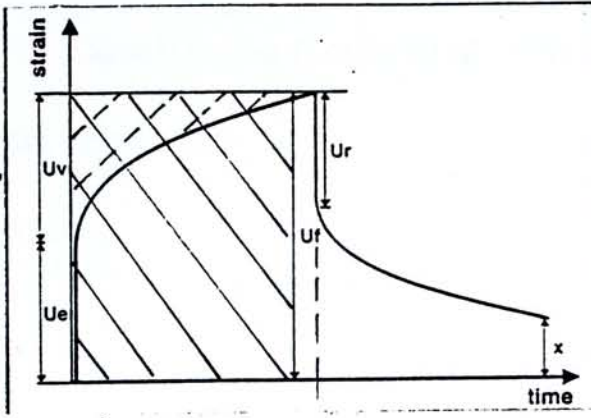
$R_6 = U_v/U_e$, the biologic elasticity, the ratio between the viscoelastic properties of the skin and immediate distension (elastic component),

R_8 is the visco-elastic character, the shaded area above the curve in relation to the strain-time curve

The value of the ratio U_r/U_f and U_r/U_e were regarded as parallel with each other.

According to the information provided by the manufacturer, R_8 can be described with the aid of the following graph (Fig. 37).

Fig. 37



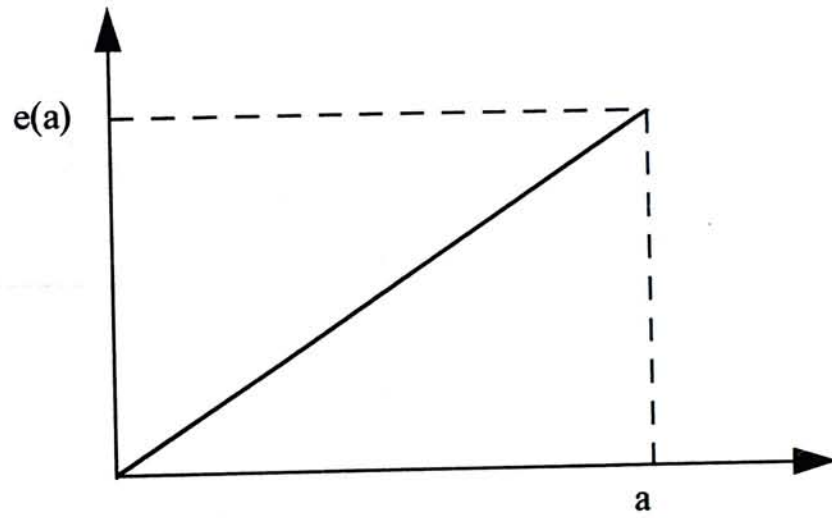
a and e(a) are marked on the drawing.

$e(a) * a * 100$ is the surface of the square.

$f(a)$ is the surface below the curve.

When looking at a curve

as follows (Fig. 38),



the relation between the surface of the square and the surface below the curve is 2:1.

For the reason, the formula is multiplied with 2 :

$$R8 = [e(a) * a * 100 / f(a) - 1] * 2$$

Hence the thickness of scar tissue, the data R0, R2, R5, R6 and R8 from longitudinal follow up of the hypertrophic scar and the corresponding normal skin on the contralateral side of the body will be taken and being analysed.

If the hypertrophic scar becomes mature, and suspension of treatment is indicated, at least one follow up measurement will be taken when patient stop therapy.

6 **CLINICAL RATING SCALE**

Clinical rating (Leung et al, 1984) summarized in Table 4. was kept as usual for comparison.

Table 4

Grade	Scar Assessment
0	Normal skin
1	Soft, paper thin, pink
2	Soft, thin, pink to red
3	Firm, moderate thickness, red
4	Firm, thick, red to purple
5	Hard, thick, purple

7 **STUDY OF NORMAL SKIN AS CONTROL**

The ultrasound image and elasticity of normal skin on the contra-lateral side of the corresponding lesion were taken every time as control. From January to May 1995, a total of 90 sites of normal skin were identified from another 29 patients and studied.

The 90 area of normal skin were categorized into abdomen, upper limb (arm & forearm, wrist & elbow), foot, leg, knee, and thigh. The average value of R0, R2, R5 and R8 of the normal skin will be taken as reference.

8 RELIABILITY OF THE ULTRASOUND AND CUTOMETER MEASUREMENT

A small scale reliability test was performed. 12 hypertrophic scars were selected from 3 patients. They were assessed by 3 observers, each observer rated the scar 3 times individually at an interval of 20 minutes. Pressure garments were removed and the patients were requested to rest in a room with temperature about 22 - 24 °C at least 30 minutes before the assessment.

108 measurements were taken from the 12 scars. Then the cutometer and ultrasound reading were analyzed by 2-way ANOVA with repeated measurements. The correlation coefficients for thickness and the maximum excursion of skin under constant pressure (R0) measured by individual examiner would be analysed with 95% confidence level. In order to eliminate the interaction-effect, the variation of various sites were also analysed. The analysis was based on the assumption that individual scars behave uniquely. Though scars were assessed from the same patient, they may be treated as individual sample.

In addition, the within-subject effects of examiner and scar would be analysed respectively. Hence, the result would reveal corresponding intra-examiner variability of the measurement of ultrasonography and elastometry.

992 valid values of the hypertrophic scars concerning the clinical grading were collected and analysed. This clinical observation included the change of the appearance, firmness/ pliability, height of scar above normal skin, and the vascularity.

817 and 992 valid values concerning the corresponding ultrasonic measurement and elasticity properties were analysed and compared with the clinical grading

Chapter Five : RESULTS

Hypertrophic scarring is assessed clinically by the change of the appearance, firmness/ pliability, height of scar above normal skin, and the vascularity. The ultrasonographic measurement and elastic properties of the hypertrophic scar are analysed and compared with the clinical grading.

1 INTER- AND INTRA- EXAMINER VARIATION OF THE
ULTRASOUND AND CUTOMETER MEASUREMENT

108 measurements were taken from 12 scars of 3 patients. The measured thickness (ultrasonography) and elasticity R0 (elastometry) were compared with the corresponding clinical observation. In order to maximize the accuracy of measurements, the data were regarded as repeated measurements and analysed by 2-way ANOVA.

Table 5

	Thickness	Elasticity
correlation coefficient	0.930	0.776
Significance of F (among scars)	0.000	0.000
Significance of F (among examiners)	0.539	0.044

Measured scar thickness and observed scar height

The correlation coefficient between measured and estimated thickness is 0.93. The Significance of F among scars and examiners were 0.000 and 0.539 respectively.

The result implies that the measured thickness correlate well positively with the estimated height. There is a significant variation from scar to scar. However, the statistical significance does not support the difference among examiners. This implies there is no significant difference among examiners in the use of ultrasonography to measure the thickness of hypertrophic scar.

Elasticity R0 and estimated firmness

The correlation coefficient for elasticity with estimated firmness is 0.776. The Significance of F among various scars and examiners were 0.000 and 0.044 respectively. The result implies that R0 correlate positively with the estimated firmness. However, there is significant difference among the scars and examiners.

Intra-examiner variability of the 2 measurements

The within-subject effects concerning thickness and elasticity are as following:

Table 6

significance of F for the within-subject effect	Thickness	Elasticity
"intra-scar" variability	0.095	0.238
"intra-examiner" variability	0.703	0.351

Concerning the measurement of thickness, both values of significance of F are > 0.05 . There is no significant difference of repeated measurement within individual scar and examiner respectively. However, the "intra-scar" variation

($p = 0.095$) is relatively significant than the "intra-examiner" variation ($p = 0.703$) in the assessment of hypertrophic scar with ultrasonography.

In the measurement of elasticity (R0), both values of significance of F are > 0.05 . There is no significant variation within individual scar and examiner.

2 COMPARISON WITH NORMAL SKIN CONTROL

90 different area of normal skin from 29 patients were studied. Different area were categorized into abdomen, upper limb (arm & forearm, wrist & elbow), foot, leg, knee, and thigh. The reading were summarized as in Table 7.

Table 7 Average reading of normal skin from cutometer were categorized in accordance with different body parts. (After Fong et al 1995, pending published in BURNS)

Area	no. of patient	no. of area being assessed	R0	R2	R5	R8
abdomen	4	4	0.438	0.954	1.005	0.105
Upper limb	9	19	0.496	0.955	0.997	0.106
arm & forearm	(8)	(14)	0.516	0.958	1.010	0.106
wrist & elbow	(1)	(5)	0.442	0.944	0.961	0.106
foot	7	11	0.328	0.936	0.952	0.169
leg	7	8	0.278	0.910	0.939	0.135
knee	5	8	0.329	0.934	0.971	0.118
thigh	15	27	0.439	0.962	1.017	0.098
Total	29	90				
Average			0.420	0.949	0.996	0.116

The average of values R0, R2, R5 and R8 were taken as reference. Normal skin usually gave a visco-elastic character (R8) around 0.1. Both Gross-elasticity

(R2) and Net-elasticity (R5) around 1, but varied for maximum excursion under constant pressure (R0) among different body parts.

From the data gathered from the normal skin, certain areas tended to give a lower elasticity (R0, R2 & R5). These areas included knee, foot and leg as the lowest. The elastic properties of the elbows were lower than the arm or forearm. On the other side, the visco-elastic property (R8) was greatest at the foot, then the leg.

3 *RESULTS OF ULTRASONOGRAPHIC MEASUREMENTS OF HYPERTROPHIC SCAR & ITS CORRELATION WITH CLINICAL GRADING*

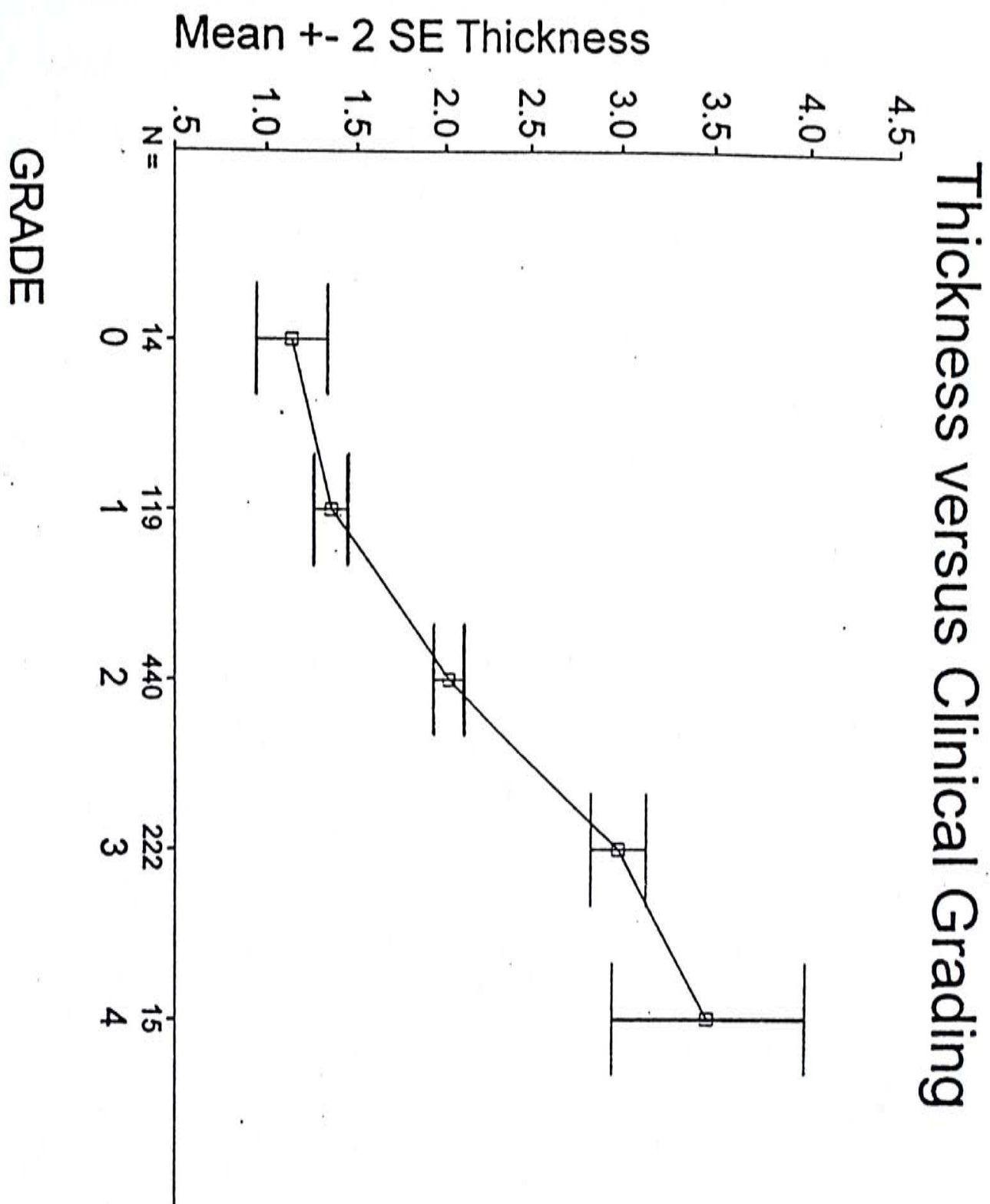
The mean value of the ultrasonographically measured thickness of hypertrophic scar was found to bear a positive correlation with the clinical grading. That is, with higher clinical grading, the mean value of the thickness increased. The correlation coefficient computed with the non-parametric Spearman Sign-Rank Test was 0.52 ($p=0.000$). The relation is quite linear from grade 1 to 3 (Graph 1). While from grade 0 to 1 and 3 to 4 the gradient is more gentle.

Table 8

Clinical grading	Mean of thickness (mm) \pm SE
0	1.238 \pm 0.118
1	1.361 \pm 0.047
2	2.205 \pm 0.044
3	2.984 \pm 0.075
4	3.467 \pm 0.256

As the sample size of grade 0 and grade 4 is quite small, the standard error is higher.

Graph 1



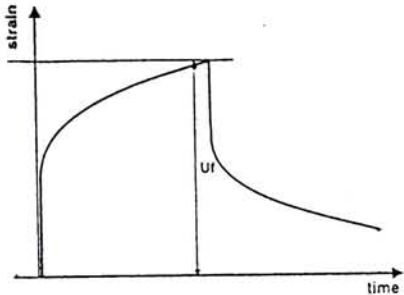
4 RESULTS OF CUTOMETER READING (VISCO-ELASTIC PROPERTIES) & THE CORRELATION WITH CLINICAL GRADING

The results were analysed with the Spearman Sign-Rank Test. The correlation coefficients for each visco-elastic property obtained with 2-tail significance were tabled respectively (Table 9) :

Table 9

	Spearson correlation coefficient	p-value
R0	- 0.4747	0.000
R2	- 0.1179	0.000
R5	- 0.1325	0.000
R6	0.3990	0.000
R8	0.3694	0.000

R0 The total deformation of the skin Uf (graph)



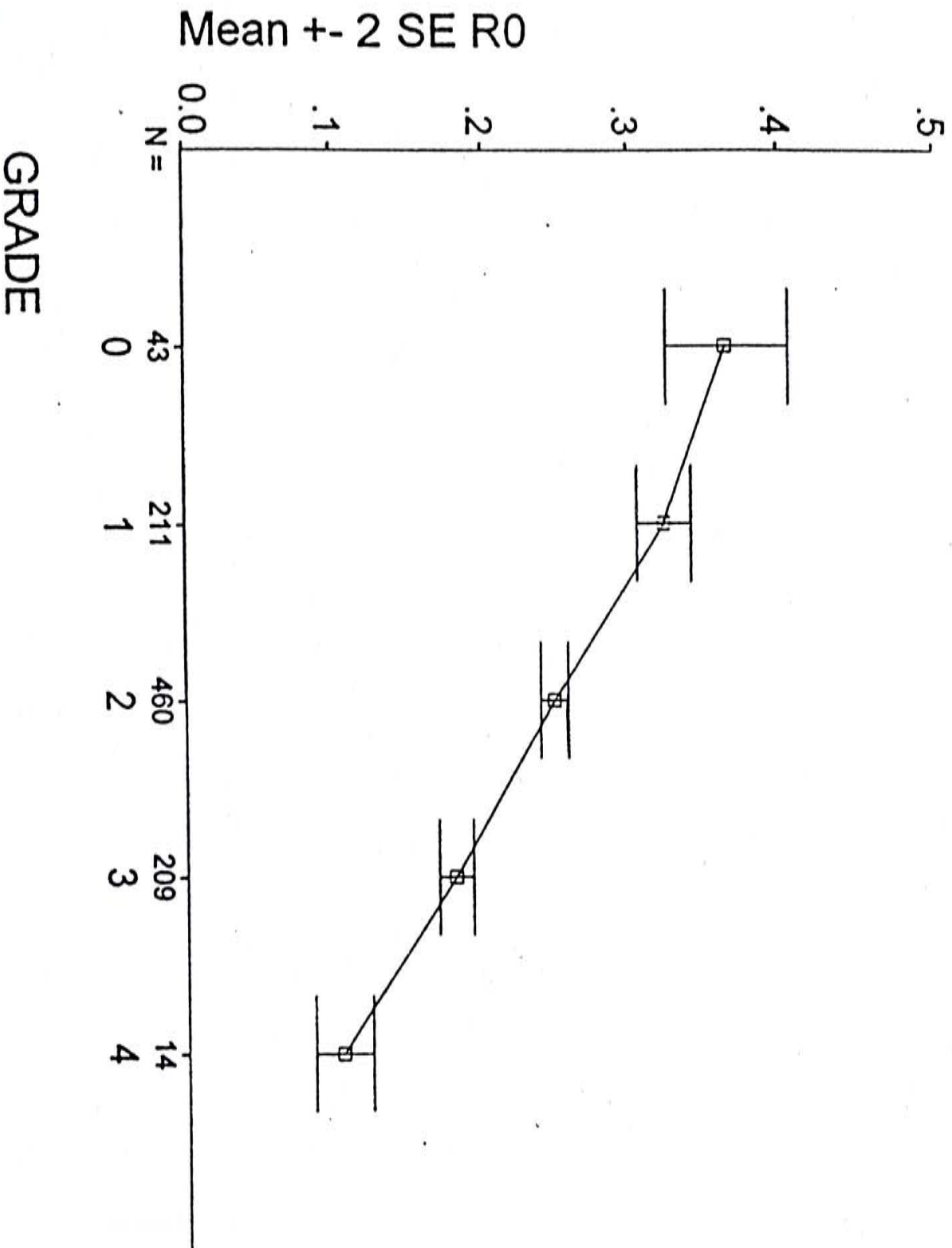
The mean value of R0 showed a negative correlation (Spearman correlation coefficient -0.4747, p= 0.000) with clinical grading. This implied that there was a reduction of excursion of skin in response to constant pressure with higher clinical grading.

Table 10

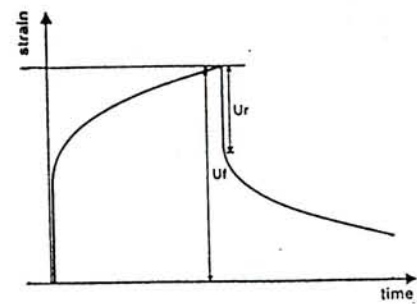
Clinical grading	Mean of R0 ± SE
0	0.367 ± 0.020
1	0.325 ± 0.009
2	0.248 ± 0.005
3	0.181 ± 0.006
4	0.106 ± 0.010

A true linear correlation of R0 with the scar was obtained from grade 1 to 4.
(Graph 2)

Mean RO vs Grading



R2 The gross elasticity, U_r/U_f . The ratio between immediate retraction and total distension representing the skin's ability to recover to its initial position after deformation.



The mean values of R2 of hypertrophic scar was much lower than normal skin. Normal skin has elasticity R2 value close to 1. (affected by aging, trauma, etc.) Both matured and active scar tissues showed reduced R2 value.

No consistent association between mean value of R2 and clinical grading was found. There is no significant statistical data (Spearman correlation coefficient is -0.1179) to support the association of R2 with clinical grading (Graph 3).

Table 11

Clinical grading	Mean of R2 \pm SE
0	0.852 \pm 0.018
1	0.847 \pm 0.009
2	0.802 \pm 0.006
3	0.820 \pm 0.008
4	0.780 \pm 0.021

R5 The net elasticity, U_r/U_e , the ratio of the immediate retraction to the immediate distension. This ratio is considered as a biological important factor for the characterization of elasticity of the skin.

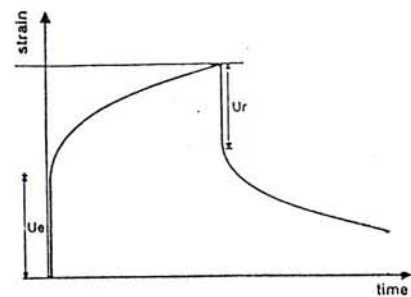


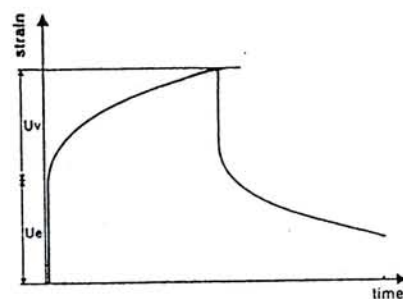
Table 12

Clinical grading	Mean of R5 \pm SE
0	0.778 \pm 0.024
1	0.769 \pm 0.014
2	0.693 \pm 0.011
3	0.729 \pm 0.017
4	0.734 \pm 0.040

The mean value of R5 for normal skin is around 1.0, while for hypertrophic scar, it was found to be around 0.85 or less. Hypertrophic scarring has a smaller value of R5 than normal skin.

There is no significant correlation between the mean of R5 value and clinical grading (Spearman correlation coefficient is -0.1325), similar to R2, the net elasticity (Graph 4).

R6 The biologic elasticity, U_v/U_e , the ratio between the viscoelastic properties of the skin and immediate distension (elastic component).



There was a positive association between the mean value of R6 and clinical grading from grade 0 to 3. The value of viscoelastic properties increase dramatically for the highest clinical graded scar, Grade 3 to 4 (Graph 5). The Spearman correlation coefficient for R6 with Clinical Grading is 0.3990, (with $p = 0.000$). The statistical data supported the positive correlation between R6 value and Clinical Grading.

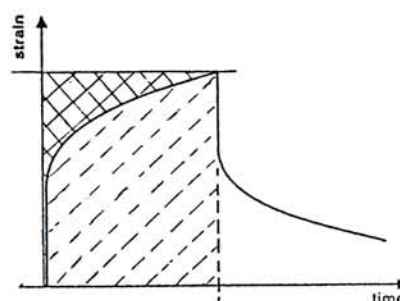
Table 13

Clinical grading	Mean of R6 \pm SE
0	0.191 \pm 0.012
1	0.230 \pm 0.008
2	0.292 \pm 0.006
3	0.357 \pm 0.010
4	0.497 \pm 0.061

The mean of R6 for normal skin is around 0.15 and hypertrophic scar has greater than 0.20 noted.

The value of R6 (> 0.377) may serve as a cut-off point between milder and very firm scar with 95% confidence level.

R8 The visco-elastic character, the shaded area above the curve in relation to the strain-time curve.



The mean value of R8 for normal skin was around 0.10, but > 0.10 in general for hypertrophic scar.

Table 14

Clinical grading	Mean of R8 \pm SE
0	0.118 \pm 0.007
1	0.135 \pm 0.004
2	0.166 \pm 0.003
3	0.181 \pm 0.006
4	0.276 \pm 0.023

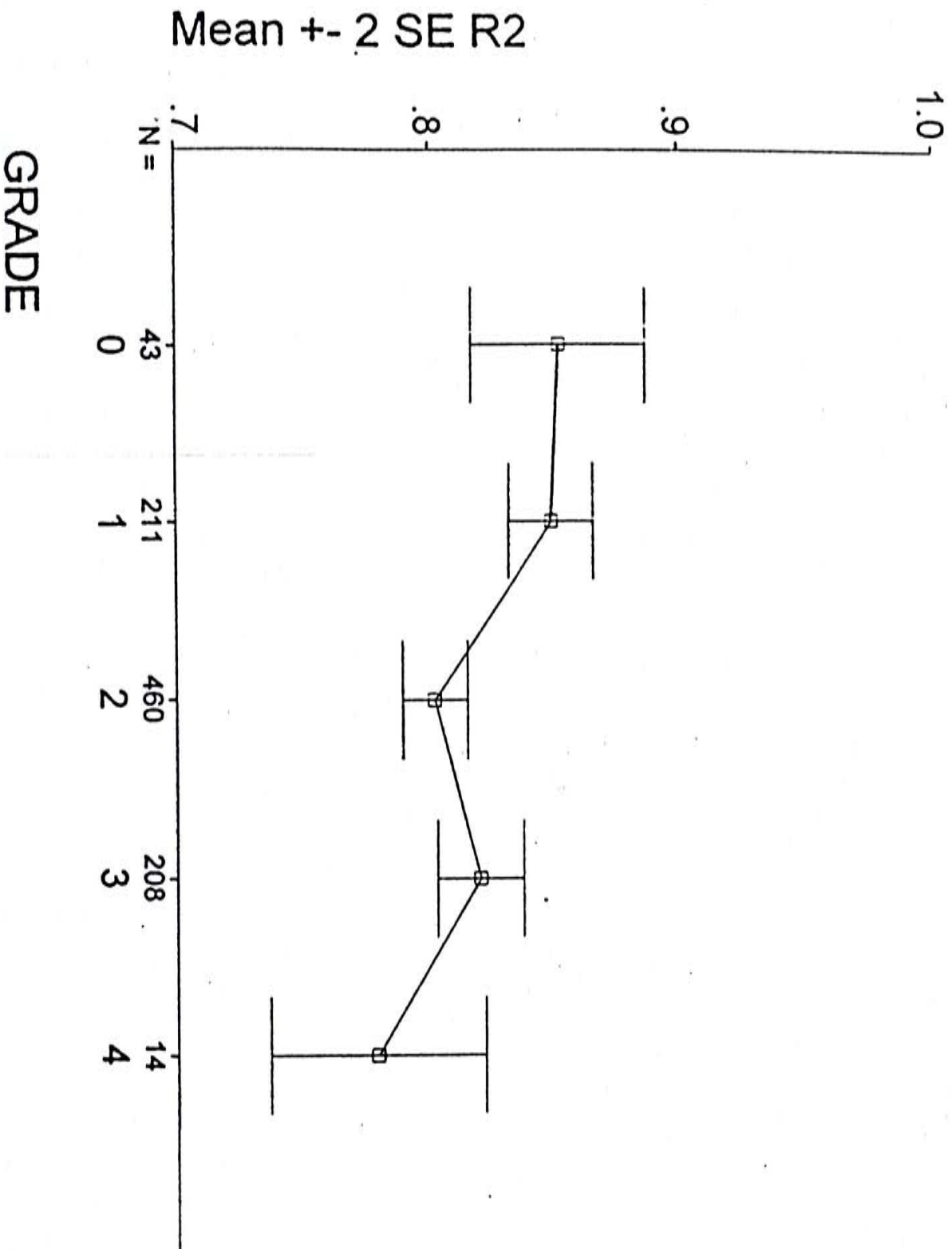
The Spearman correlation coefficient of R8 to the Clinical Grading is 0.3694. With 95% confidence level, the statistics support the positive correlation of R8 to Clinical Grading.

As noted in comparing mean value of R6, the viscosity (R8) of hypertrophic scar increase gently for scar with clinical grading from 0 to 3, and more drastically for grade 4 (Graph 6).

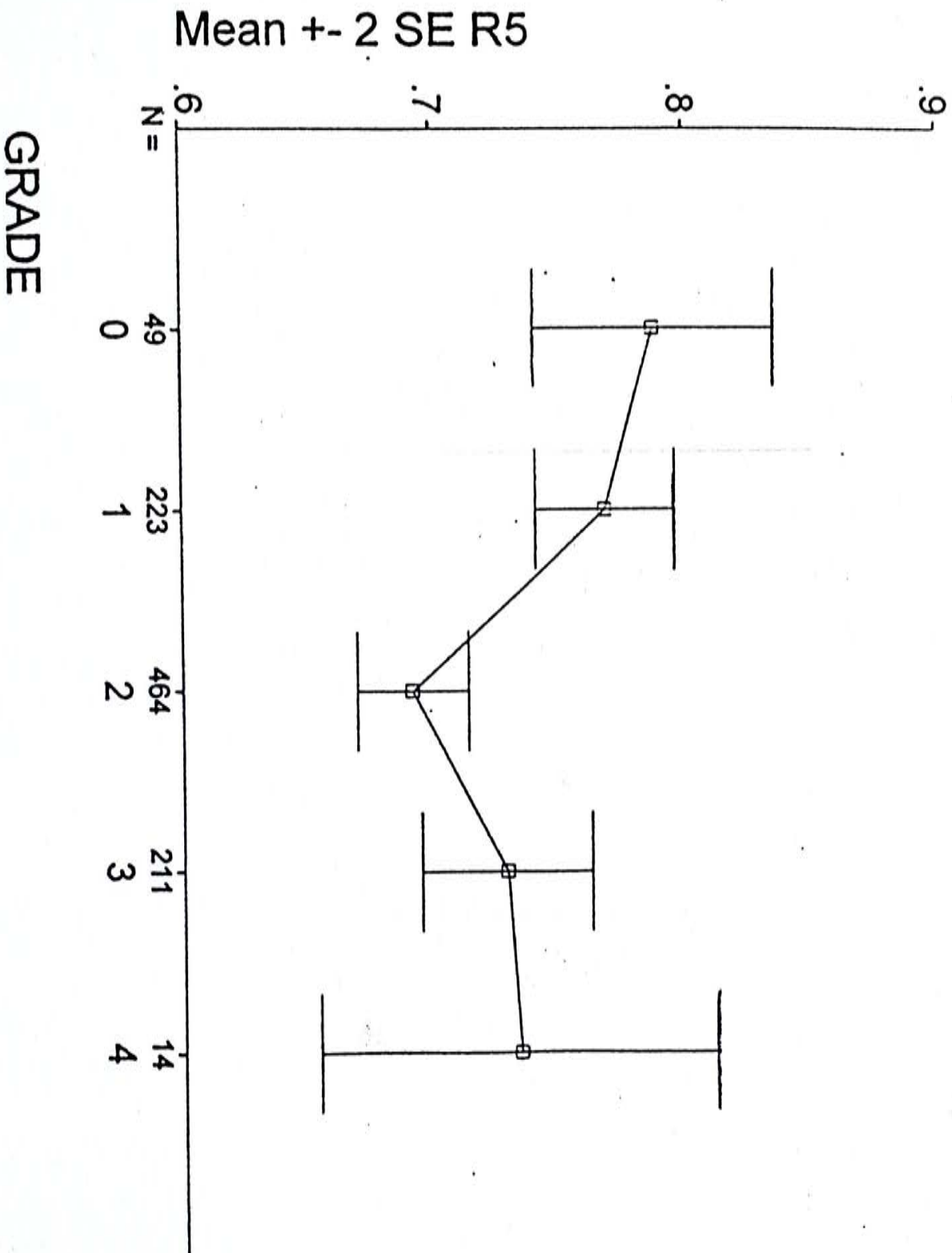
The value of R8 greater than 0.193 may serve as a cut off point between grade 3 and 4 scar of 95% confidence.

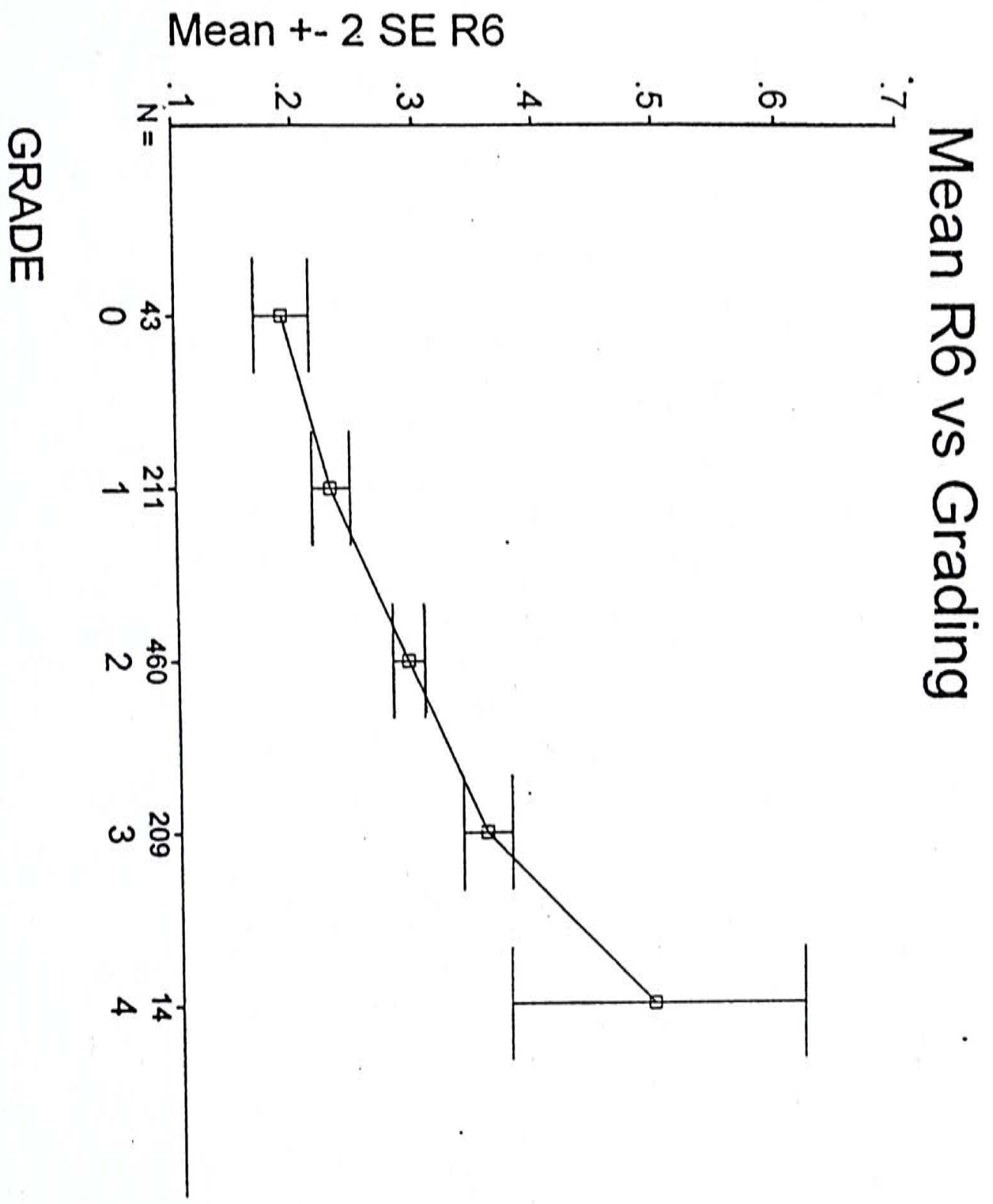
The dramatic increase in slope may be affected by the greater standard error of the small sample size ($N=14$).

Mean R2 vs Grading

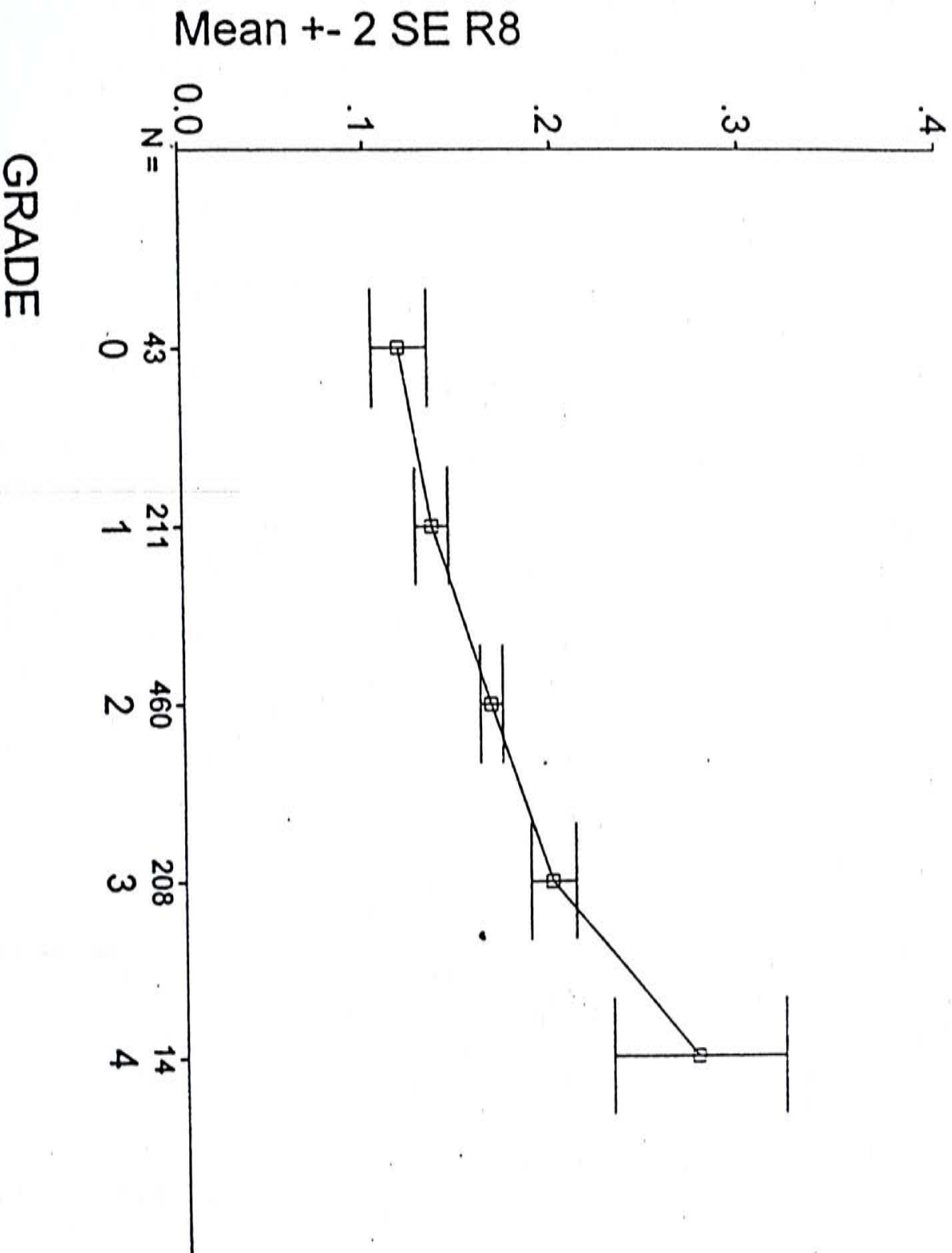


Mean R5 vs Grading





Mean R8 vs Grading



5 OBSERVATIONS FROM RAW DATA

R0

1. For active scar, the R0 value laged far behind normal skin.

The ratio of $[R0(HS) - R0]/R0$ was negative in general.

2. As scar matured, there was a general increase in R0 with time in response to constant pressure, but the changes seldom exceed beyond value of normal skin.
3. For very firm scar tissue, the strain-time curve shows a very different pattern from normal skin, and the skin will retract immediately to zero after releasing the suction force.
4. Fresh scar will resemble normal skin value faster.
5. Less fluctuation was noted for chronic scar (more than 12 months). On the contrary, greater fluctuation can be observed in fresh scar.
6. With SSG, it took shorter time to reach a higher distention.
7. With injection, the fluctuation of the curve increase, skin distention due to constant pressure increased dramatically with 1 to 2 months time, then gradually decline 4 to 5 months after injection therapy and having similar modulus as pre-injection state or with mild improvement. The sudden increase in the excursion coincised with the noted atrophic change of the epidermis.
8. A different Young's modulus is noted at different sites even in normal skin. In general, tissue like groin area, thigh, abdomen with more fascia and fatty tissue will have increased Young's modulus. However, the scar at these sites will show a longer and more fluctuating R0 value.

R2 and R5

1. No linear nor exponential curve can be drawn from hypertrophic scar. This may reflect a constant, dynamic change of elasticity during scar tissue remodelling.
2. R2 and R5 value of normal skin for all sites was approximately equal to 1.0.
3. For very thick and firm scar, R2 value is greater than 1. This can easily be explained with the strain-time curve. At removal of the suction, the curve will decline to zero without delay, representing a very high stiffness of the scar tissue.

R6

1. Higher clinical graded hypertrophic scar has a greater value of R6 than normal skin.
2. Active scar will have a higher ratio of $[(HSR6 - NSR6)/NSR6]$
3. R6 will increase 4/12 after injection.

R8

1. R8 for normal skin was approximately equal to 0.1 for all body parts.
2. A greater R8 value was shown with different grading of hypertrophic scar.
3. R8 value increased drastically for Grade 4 clinical scar.
4. As scar become mature, the value of R8 will decrease and approaching the value of normal skin.
5. Injection of topical steroid will induce skin atrophy and value of R8 reduce dramatically. During longitudinal follow up, this will occur 1 to 2 months after injection. However, the visco-elasticity R8 will gradually increase to a lower level 4 to 5 months after the injection.

Chapter Six : DISCUSSION

Hypertrophic scar is a consequence of an abnormal wound healing process. The depth and the extent of the injury affects the development and severity of hypertrophic scar. In addition, site of wound and races will also affect the severity of scarring. It is very common to find hypertrophic scar from split-thickness donor site in the Asian population.

Hypertrophic scar is thick, firm with reduced elasticity and highly vascularized. Patients with hypertrophic scars suffer from persistent itchiness and often irritating pain. If the scar stretched across joints, it will develop into joint contractures and deformities that could limit the range of motion and finally hinder the functions of limbs or other body segments.

Eventually, the hypertrophic scar will become mature and softens with time though rarely ever return to normal fully. This will last for weeks to years. With the advancement in technology and clinical research, medical staff can use different methods to promote wound healing and hence reduce the possibility of developing into severe hypertrophic scar.

In the past 20 years, several studies have been carried out to verify the effectiveness of different treatment modalities of hypertrophic scar. (Ketchum & Cohen 1974, Longacre et al 1976, Lehmann et al 1983, Kischer 1975, Kischer et al 1978, Larson et al 1973, Quinn 1985, Cheng et al 1987.)

The external appearance and mechanical properties are generally accepted as assessment method to document the changes and remodeling of scar since it is both non-invasive and do not require any equipments.

Most studies are short terms (less than 6 months), just long enough to determine if the treatment is effective. Yet, few of them has validated the effectiveness. Katz et al (1985) tried to study the use of ultrasonography and tonometry in the assessment of hypertrophic scar over 2 month's time. The ultrasonography and tonometry were concluded as reliable in the measurement of hypertrophic scar. However, it did not tell the longitudinal changes of the morphology of scar tissue.

Naismith (1980), Hambleton & Shakespare (1987) and Clark et al (1987) conducted longitudinal follow-up studies (more than 12 months). None of these can substantiate the change of scar as a function of time. Most of the studies are laboratory-based and too tedious for regular clinical measurement.

The widely used Vancouver Scar Scale/ Burn Scar Index (Sullivan et al 1990) is based on the clinical grading of the appearance of scar (including pliability, scar height above skin, vascularization, pigmentation). The items are not equally weighted. It is still a subjective assessment during the grading though satisfactory inter-examiner reliability is proven (Baryza & Baryza 1995). The thickness of hypertrophic scar tends to be underestimated when compared with the real thickness of the dermal layer involved. It is revealed under the ultrasonograph. The image of a scar tissue is pickled

in shape and extends to occupy the whole thickness of the dermis that is not just raised above normal skin. (fig. 34 ultrasound image) If it is excised in situ, the scar tissue is much thicker than expected. With reference to the literature review of pathogenesis (Kischer & Brody 1981), remodeling of hypertrophic scar involves revascularization of a wound. It possesses a heavier concentration of occluded microvessels encompassing the collagen nodule and favors the subsequent proliferation of fibroblasts. Eventually the process becomes self-limiting as the hypoxia progresses to a point which result in cell death within the nodules. Hydrolytic enzymes are being released and induce the scar resolution. As the vascularization, thickness and firmness changes simultaneously, an overall summation of the scores as in the Vancouver Burn Scar Assessment Scale will magnify the change.

As there is no consensus in the triggering factors or the detail pathogenesis of hypertrophic scar. Treatments are still limited to "treating the symptoms". Currently, pressure therapy, topical silicone gel, intralesional injection of cortico-steroid, and surgery or combination of the above-mentioned is widely used. Effect of new modalities such as laser treatment is under investigation.

Objective and standardized assessment tool(s) is/are essential to quantify and document the change of hypertrophic scar. This would allow more precise monitoring of the progress of hypertrophic scar under different treatment modalities.

In this study, ultrasonography and elastometry were adopted to assess the thickness and elastic properties of hypertrophic scar longitudinally. The result was

compared with clinical grading; the predictability and reliability of the assessment tools were analysed and discussed.

1 MEASURING THICKNESS WITH ULTRASONOGRAPHY AND CLINICAL GRADING

The measured thickness of hypertrophic scar with ultrasonography was found to correlate well with the clinical grading. The correlation coefficient is 0.520 with $p=0.000$, variation is less than 5% significance. The scar were thicker with higher clinical grading. The relation is quite linear from grade 1 to 3. From grade 0 to 1 and 3 to 4 the gradient is more gentle. (see Graph 1, p.141)

The predictive value of measured thickness of scar is reliable in the assessment of hypertrophic scar. The sample size of grade 0 and grade 4 is too small, hence a greater standard error is obtained which has affected the linear continuity at both ends. In modern clinical practice, early interventions to provide good wound dressing such as artificial dressing, allograft and autograft would be provided. This will shorten the wound healing time and lessen the chance of developing active, hard scar and contractures. Therefore, a grade 4 and even higher graded scar is less common and this contribute to the small sample size. On the other side, patients with normal scar grade 0 will either drop out or not being referred to the specialist clinic. This is also one of the reason that leads to the small sample size. Hence the result can be biased.

2 ELASTIC PROPERTIES OF HYPERTROPHIC SCAR AND CLINICAL GRADING

The mean value of R0 showed a negative correlation (- 0.4747, $p=0.000$) with clinical grading. That implies that there was a reduction of excursion of scar with higher clinical grading.

There was no consistent association between mean value of R2 and R5 with the clinical grading. The correlation coefficient of the R2 and R5 versus Clinical Grading were -0.1179 and -0.1325 respectively. Probably due to the fact that clinical grading is subjectively not sensitive enough to be certain in differentiating minor to moderate changes in color and firmness during scoring. Hence, statistically, R2 and R5 values did not correlate well with clinical grading.

In addition, the fluctuating reading of R2 and R5 matched with the dynamic remodeling phase of scar tissue (Kischer 1982), i.e. synthesis of collagen balanced out with its degradation.

Clark et al (1987) monitored the progress of post-burn hypertrophic scar by measuring the mechanical properties, the modulus of elasticity (E) and strain (ϵ) at different load intensities. These mechanical properties correlate with clinical assessment of hypertrophic scar grading. Higher scar grading is indicative of increased stiffness (Modulus of Elasticity) and decreased extensibility. However, the result obtained by the values of R2 and R5 do not agree with this observation (Graph 3, p.148 and Graph 4, p.149). So, a difference is obvious between

stretching and suctions device. Stark's hypothesized (1977) the time-dependent initial behavior of collagen content in response to stretching (as reviewed in the mechanical properties of skin). This can also help to explain the change in visco-elastic properties of hypertrophic scar under constant suction.

The positive and linear association was reflected between the mean value of R6 and clinical grading from grade 0 to 3, and accelerated from grade 3 to 4 (Graph 5, p.150). The value of viscoelastic properties increased dramatically for highest clinical graded scar. The cut off point at grade 3, with mean of R6 greater than 0.377 may separate very firm scar from less severe ones and may indicate that additional intervention is necessary.

The mean value of R8 similar to R6, increased gently for scars with clinical grading from 0 to 3, and more drastically for grade 4 (Graph 6, p.151). The value of R8 greater than 0.193 (mean value of R8 at grade 3 + 2SE) may serve as a cut off point at grade 3 and can separate the hard scar with high viscous properties from less severe ones.

Both R6 and R8 values describe the visco-elastic properties of hypertrophic scar and confirmed the higher viscosity of hypertrophic scar than normal skin. With higher clinical grading, scars are regarded as firmer, thicker and increased in vascularity. The elastic content is hence much lower resulting in the increased in resistance to stretching. With a higher visco-elastic property and reduction in the distention, the shape of the strain-time curve in response to suction shift

downward. Therefore, the corresponding R6 and R8 value rose in comparison with normal skin.

The small sample size (N=14) with a greater standard error caused the dramatic increase in slope of R6 and R8. As being discussed, there were fewer cases with higher clinical grading due to the early intervention treatment.

In the study of normal skin, certain body regions with more subcutaneous tissue such as thigh, abdomen, arm and forearm, will give a greater value of R0, R2 and R5 than the average values. While other areas with bony prominence underneath, such as foot and elbow tends to have a lower value of the elastic components. Among R0, R2 and R5, the variation of R0 values are greatest for different body regions.

The average of visco-elastic property (R8) of normal skin is quite consistent except it was highest at the foot.

All the evidences suggested a great variation of R0 for different body parts. Regional differences in mechanical characteristics adapt skin to local demands. This also reflect the local structure of the dermal collagen and elastic networks (Montagna & Parakkal 1974).

The value of R2, R5 and R8 of normal skin suggested a more stable level for reference.

3 THE PREDICTIVE VALUE OF THE ULTRASONOGRAPHY AND ELASTOMETRY THROUGH MONTHLY LONGITUDINAL MEASUREMENT

Fong et al (1995) had conducted a preliminary study of 62 scars in 5 months to study the use of cutometer and ultrasonography in the assessment of hypertrophic scar. The cutometer and the ultrasound imaging are more sensitive and specific than that subjective clinical rating scale.

Table 15 Summary of different measurements of hypertrophic scars
(After Fong et al 1995, pending published in *BURNS*)

Result	Sonograph reading	Cutometer				Clinical rating	
		R0	R2	R5	R8	colour	consistency
increase	13(21%)	45(75%)	28(47%)	20(33%)	29(48%)	0(0%)	2(3%)
decrease	24(39%)	14(23%)	29(48%)	39(65%)	31(52%)	24(39%)	21(34%)
no change	25(40%)	1(5%)	3(2%)	1(2%)	0(0%)	38(61%)	39(63%)
Total	62	60	60	60	60	62	62

Table 13. Elasticity of hypertrophic scar and the ultrasonographic presentation.
(After Fong et al 1995, pending published in *BURNS*)

Sonograph	no.		R0	R2	R5	R8
↓ thickness	23	range	↓32% -↑225%	↓17% -↑25%	↓33% -↑44%	↓62% -↑126%
		average	↑ 56.9%	↑ 0.26%	↑ 6.5%	↓ 5.7%
no change	24	range	↓42% -↑200%	↓53% -↑21%	↓57% -↑65%	↓61% -↑273%
		average	↑ 36.2%	↓ 5.8%	↓ 6.3%	↑ 10.9%
↑ thickness	11	range	↓110% -↑ 188%	↓13% - ↑29%	↓48% -↑44%	↓63% -↑65%
		average	↑ 55.3%	↑ 9.1%	↑ 1.5%	↓ 0.3%

Both the cutometer and the ultrasonograph imaging are more sensitive and specific than the subjective clinical rating scale that is illustrated in Tables 15 & 16. 61% and 63% of the hypertrophic scar were graded as no change in color and consistency, whereas only 40% of the measurement by ultrasound agree, and cutometer measurement never have consistent result. Accuracy of the ultrasonograph imaging measures up to 0.10 cm and 0.010 mm for cutometer.

The predictive values of R0 in a longitudinal follow up was reliable. A true linear correlation of R0 with the injured skin was obtained especially from grade 1 to 4. In addition, a relatively more gentle change from grade 0 to 1 was noted and the standard error in grade 0 and 4 was slightly higher because of the little sample size. Nevertheless, R0 value is recommended for the documentation and monitoring the progress of hypertrophic scar.

R2 and R5 of both active and mature scar tissues was generally lower than normal skin. On the other hand, the fluctuation of the elastic properties (R2 and R5) is noted with longitudinal follow up. Greenhalgh et al (1994) described the collagen content entered a dynamic balance where collagen synthesis equal collagen degradation. When scar becomes mature, the wound tensile strength continues to increase, more type III collagen is present; with maturation, more of the collagen is replaced by type I. In the same time, other extracellular matrix proteins are also modified. Uttio et al (1986) revealed types I and type III collagen correspond to the tensile strength and tensile properties respectively.

The R2 and R5 values, namely gross elasticity and net elasticity accordingly, are computed basing on the ratio of immediate retraction of skin in response to pressure. These values will fluctuate with the different content of collagen fibers that contribute to the elasticity of the skin most. Although the number does not change, the dynamic balance of the generation of new collagen and degradation of the existing collagen will affect the elasticity directly. It is because the ratio of newly formed and the degraded collagen are not constant. The fluctuations of the curves cannot give a consistent predictive value.

Instead, R6 and R8 gave a more better predictive value of grading of scar tissue. The correlation coefficients of R6 and R8 versus Clinical Grading were 0.399 and 0.3694 respectively. These two values marked the visco-elastic properties of scar tissue, are regarded as independent of skin thickness (Cua et al 1990). The extracellular matrix of dermis is changed, and leads to much higher viscosity. With gradual remodeling of the tissue, the visco-elasticity of the scar reduced gradually and resembled normal skin.

The deposition of fibrin within the matrix of the granulation tissue (Kischer 1982) and the myofibroblast (Baur 1978) also contributes to the orientation of collagen fibers and the physical properties of hypertrophic scar.

4 INTER- AND INTRA- EXAMINER RELIABILITY OF THE ULTRASONOGRAPHY AND ELASTOMETRY IN THE ASSESSMENT OF POST-BURN HYPERTROPHIC SCAR

Reliability test of the sonographic and cutometer measurement suggested good reliability among examiners and intra-examiner. For the sonographic imaging (assessment of thickness of scar) the inter- examiner reliability is high ($p < 0.000$) with 95% confidence interval; while in the cutometer measurement, there is difference among the observers ($p = 0.044$). This variability is still accepted because the difference between different sites and different scars are more significant ($p < 0.000$). This implies that training is necessary so that the examiner is familiar with the use of the cutometer.

The result also reveals the insignificant variations among measurement taken by same examiner. The intra-examiner variability is convinced. The intra-examiner variation of thickness ($p = 0.703$) is less than that of elasticity R0 ($p = 0.351$).

5 THE USE OF A COMPOSITE "VISCO-ELASTICITY-THICKNESS CHART"

The results from this study would suggest that assessment of scar *thickness* and *visco-elasticity* value R8 can help in the quantitative monitoring and documentation of the changes and maturation of hypertrophic scar.

A "Visco-elasticity-Thickness" chart can supplement to the clinical grading for interpretation. The chart incorporates both R0, R8 (y-axis on the left) and the ultrasonographic measurements (y-axis on the right) in the function of time (x-axis). It also allows longitudinal recording. A cut-off line for the value $R8 = 0.193$ (mean value of R8 at grade 3 + 2 SE) is high-lighted for reference. According to the result, with 95 % confidence level, scar with $R8 > 0.193$ can be regarded as a firm scar, that close monitoring is indicated. For firm scar without improvement in either the thickness or the elasticity, other intervention such as intralesional injection of cortico-steroid, surgical release, may be indicated.

The application of the "Visco-elasticity-Thickness" chart is illustrated with several cases in the following (Graphs 7 to 14, pp. 174-181). They include fresh surgical scar, chronic scar, grafted area and injected scar.

Case 11, YYT

This infected wound with prolonged healing time ended with hypertrophic scar. In the initial 12 months, the thickness of the scar range from 1mm to 3mm; the R0 does not change much, around 0.2mm; and the R8 fluctuated around the cut-off line 0.193, it indicated that the scar remained active (Graph 7, p.174).

Fig. 39, case 11 YYT



When pressure garment was taken off at near maturation phase, the scar deteriorated shortly and gradually improved again even if pressure garment was not resumed.

Case 7, CTK

This case was complicated with renal problems. In the initial months, he defaulted treatment. The wound on left thigh did not heal until the 10th months after injury. It was very itchy, the patient scratched and lead to frequent abrasion and wound breakdown. This affected the compliance to pressure garment much. The scar was very hypertrophic though response to pressure garment was good.

On the "Visco-elasticity-Thickness" chart (Graph 8, p.175), the scar was thick with most of the time around 4 mm in thickness; the R8 value fluctuated along the cut-off line 0.193.

Fig. 40, case 7, CTK



Case 10, LYY

This scar was closely assessed when skin atrophy noted 3 months after intralesional injection of cortico-steroid. Initially, the thickness of the scar was minimal, and R8 value went downward gradually. 9 months after the injection, the scar thickened up again and the R8 value rose up and fluctuated again (Graph 9, p.176).

This implied that the effect of injection is time-dependent; or one simple injection therapy were not able to retard the progression of hypertrophic scarring.

Fig. 41, case 10, LYY



Case 3, TSK

A chronic scar with injection done. Like the previous example, the scar was assessed 2 months after injection when skin atrophy was noted.

The atrophic skin did not show significant thickness, the R0 value increased gradually and R8 value remained low. 10 months after injection, the scar returned to the original condition (Graph 10, p.177).

Fig. 42, case 3, TSK



Case 14, TSC

The wound was closed by split-thickness skin graft. The chance becoming hypertrophic was much reduced. 10 months after operation, thickness of the scar is not obvious; the R8 value fluctuated well below the cut-off value 0.193.

When pressure garment was taken off, the elasticity of skin reduced and improved slowly 2 months later (Graph 11, p.178).

Fig. 43, case 14, TSC



Case 16, YKL

Three scars were selected from this patient for comparison.

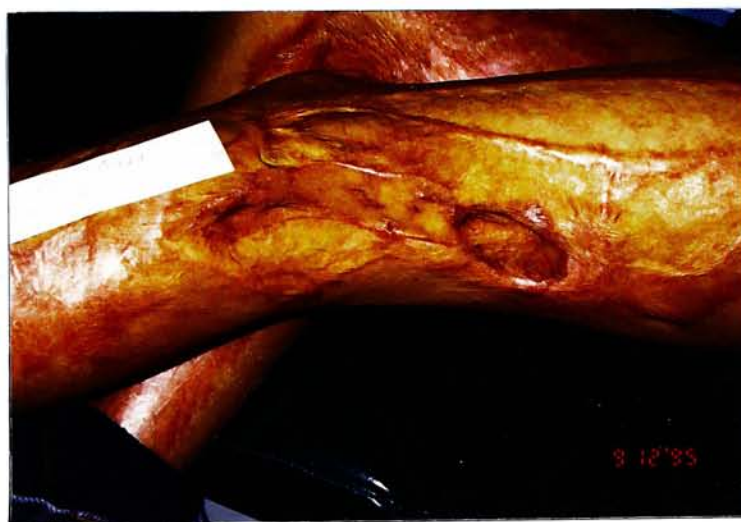
YKL suffered wax burn 59 months before initial assessment. The chronic scar on right leg was thick, R8 value fluctuated just below 0.193. The R0 changed gently (Graph 12, p.179). It was noted that 9 months after the release of the knee contracture, this chronic scar showed mild improvement in the visco-elastic characters.

Fig. 44, case 16, YKL



The scar on left lateral popliteal area was excised and covered with full thickness skin graft. The R8 recorded was around 0.193 and stayed below the value since 6 months after surgery. The corresponding measured thickness was reduced at the same time (Graph 13, p.180).

Fig. 45, case 16, YKL



The full thickness skin graft was taken from left hip and closed with a surgical scar. The thickness reached maximum at the 5th month after operation. The R0 changed progressively with a general increase of elasticity with time. R8 fluctuated about 0.193 until the 9th month after operation (Graph 14, p.181).

Fig. 46, case 16, YKL



Graph 7

Case 11, YVT
Scar 1 : Lt medial popliteal fossa, infected wound,
took one month to heal

R0/R8

1.0

0.8

0.6

0.4

0.193

0.0

Thickness
(mm)

15

10

5

0

CG

T

R8

R0

- off P.G.

- off P.G. 2/12

- off P.G. 5/12

2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19

Time (months after injury)

Graph 8

Case 7, CTK, medical history of renal problem
Scar 2 :

- wound noted on scar, delay healing, cannot measure until 10th month
- Pressure therapy suspended

- wound healed, start P.G.

- itchy⁺, abrasion noted
P.G. on and off

- P.G. stopped as pt's mother not in town
- continue off P.G. as abrasion noted
- resume P.G.

Thickness
(mm)

R0/R8

1.0

0.8

0.6

0.4

0.193

0.0

CG

R0
R8

T

3

10

11

12

13

14

15

16

17

18

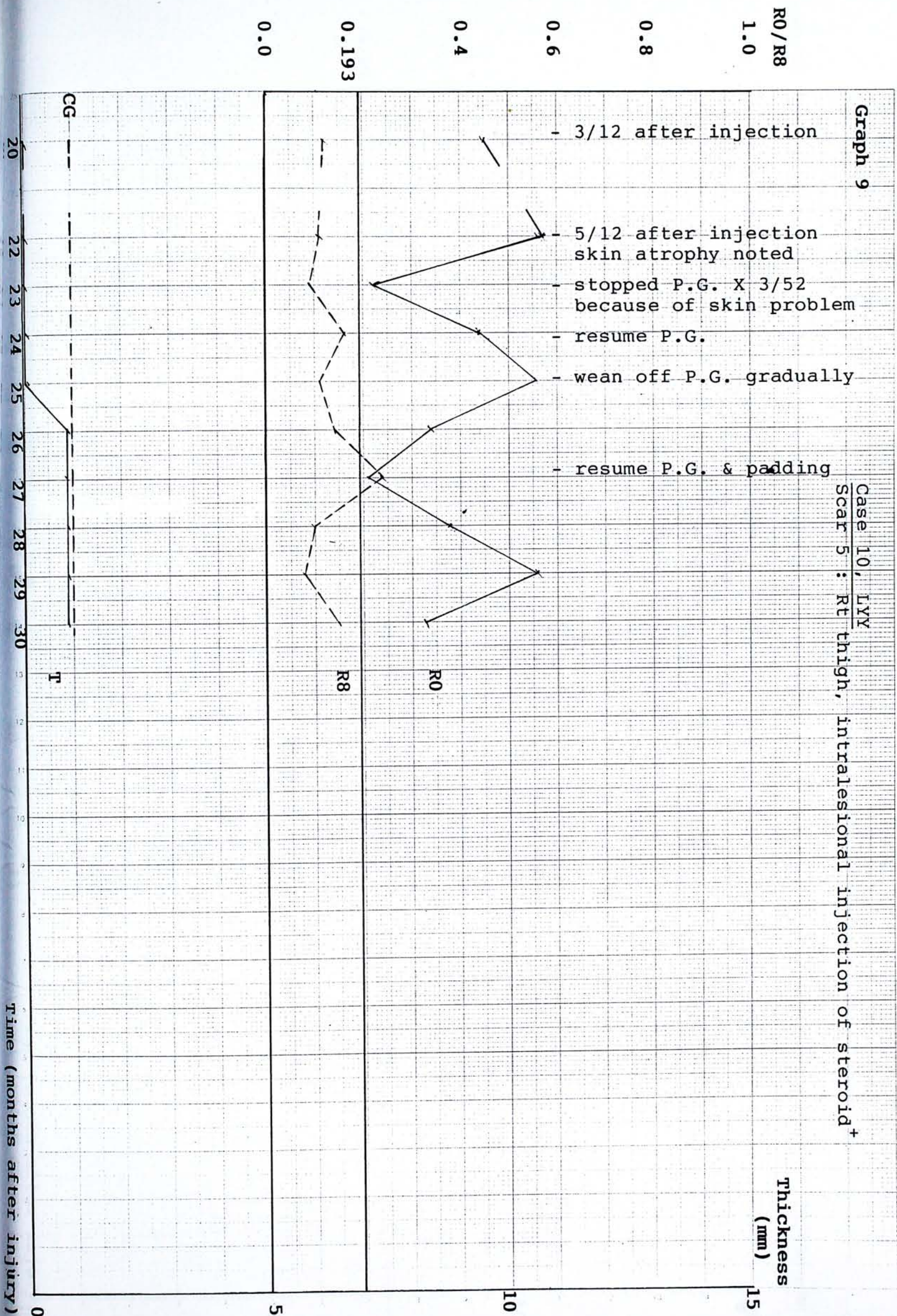
19

20

Time (months after injury)

Graph 9

Case 10, LYY
Scar 5: Rt thigh, intralesional injection of steroid⁺



Graph 10

Case 3, TSK
Scar 4 : Rt forearm, intralesional injection of corticoid steroid⁺

Thickness
(mm)

R0/R8

1.0

0.8

0.6

0.4

0.193

0.0

- 2/12 after injection, skin atrophy noted
- 3/12, skin atrophy persist, try off P.G.
- off P.G. 2/12
- stop P.G. 3/12
- stop P.G. 5/12
- stop P.G. 7/12
- stop P.G. 10/12

R0
R8

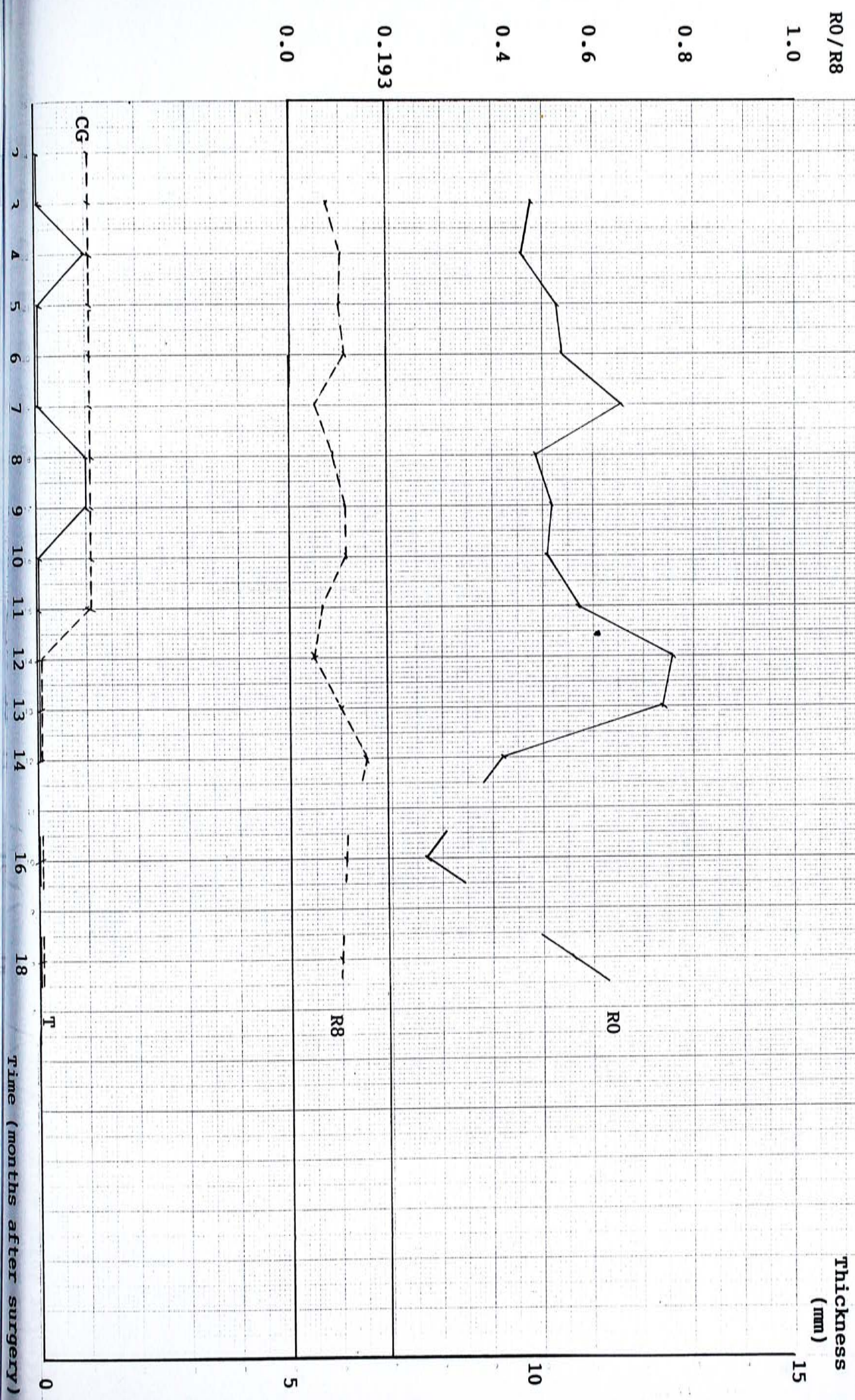
CG

T

92 96 97 98 99 100 102 104 107
2 3 4 5 6 8 10 13
Time (month after injury) 0
(month after injection)

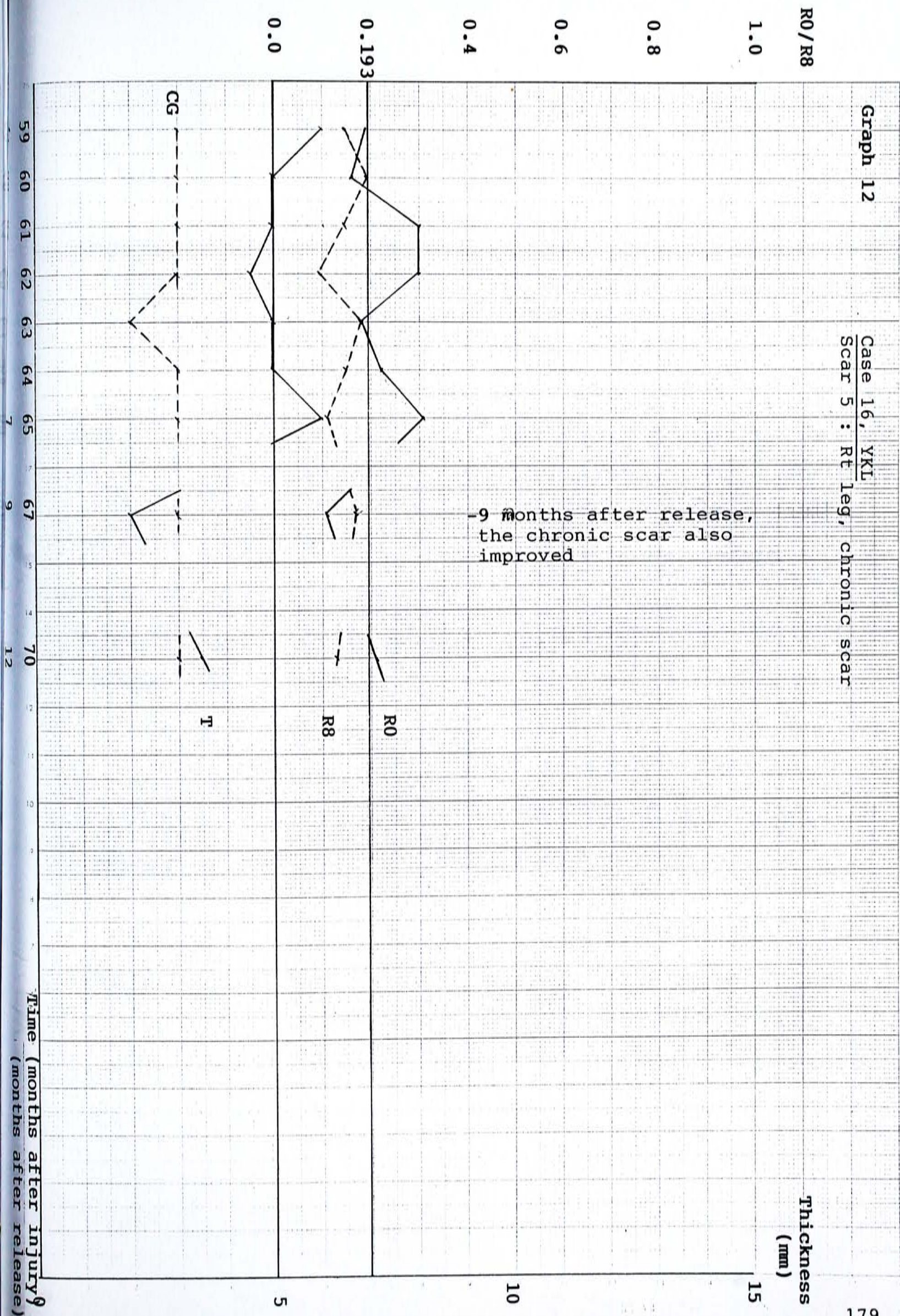
Graph 11

Case 14, TSC
Scar 3 : Rt thigh (SSG)



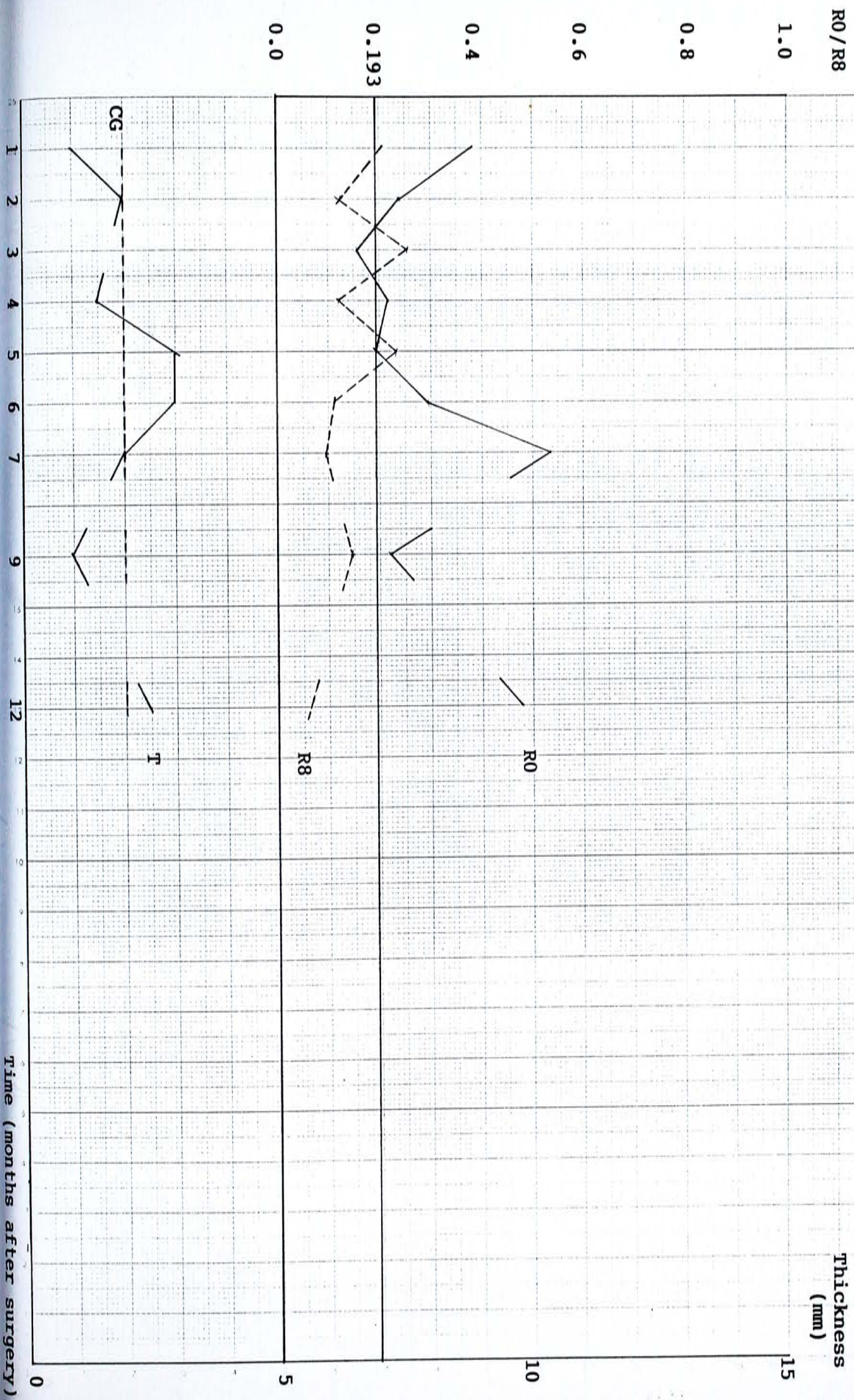
Graph 12

Case 16, YKL
Scar 5 : Rt leg, chronic scar



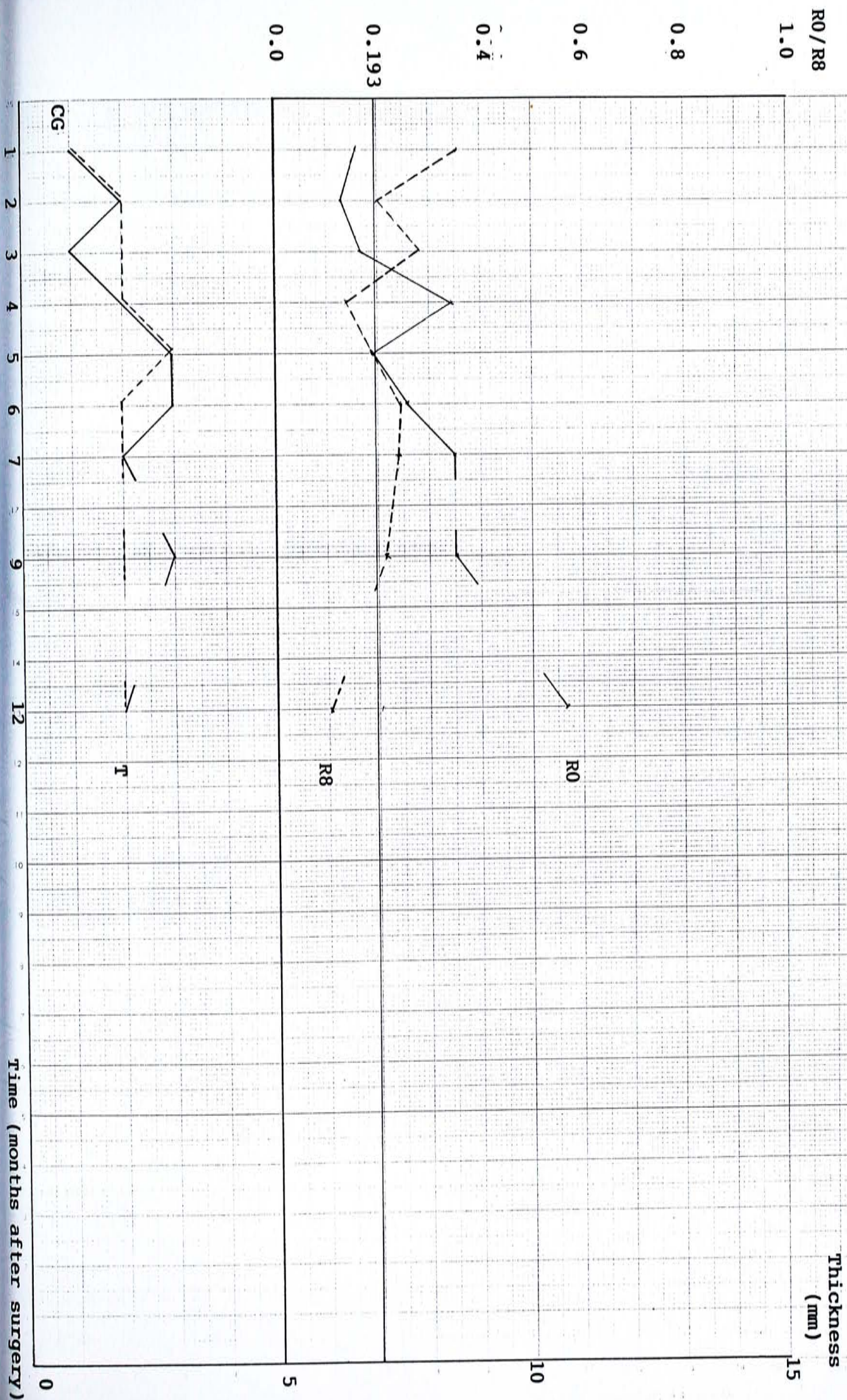
Graph 13

Case 16, YKL
Scar 3 : Lt lateral popliteal (SSG)



Graph 14

Case 16, YKL
Scar 1: Lt hip, donor site of FTSG, close with surgical scar being left



6 LIMITATIONS OF THE STUDY

Scar tissue consists of young actively growing cells, which readily *respond to changes in stress and external pressure*. Since the scar tissue is more sensitive in its early stages, it can react more favorably to appropriate corrective measures such as exercise, traction, and pressure. As the scar matures it becomes less responsive to therapy. Thus the physician should see and evaluate the patient at frequent intervals during the initial 6 months after healing (Larson et al 1971) .

The accuracy of the portable ultrasound machine (Aloka SSD 500) with the electronic linear probe (UST-5512U-7.5MHz/38mm) is only up to 0.1cm. For instance, for a 2mm hypertrophic scar, the error is up to 50%. In order to achieve a more reliable measurement, a higher frequency measurement probe with a compatible ultrasound machine are recommended. For example, ultrasound of 20MHz can give an accuracy up to 0.1mm, hence the error can be reduced significantly to 5% for a 2mm hypertrophic scar.

The cutometer is very sensitive, the reading will vary even if there is a slight movement, including that of muscle contraction. It is especially difficult in assessing children because patients always feel itching upon removal of the pressure garment. Hence resting the patients in a proper position for assessment is essential to reduce the error.

The elasticity measured would be affected by the pressure that was exerted through the measurement probe. Meanwhile, the probe does not have a system to check the pressure/ force being exerted. The probe was placed on the hypertrophic scar by its own weight. Hence, patients should be rest with the hypertrophic scars facing upward to allow a consistent perpendicular measurement. In the long run, the measurement probe should install a pressure sensor to guide the exertion of force.

Ideally, the contra-lateral limb in case of a limb burn should be used as control every time, failing that, the adjacent normal skin should be used.

The two new assessment skills are specific and reliable, but time consuming at this stage. Training is essential for the evaluator to ensure proper technique throughout assessment, and hence minimize the measurement error.

Chapter Seven : CONCLUSION & RECOMMENDATIONS FOR FURTHER STUDY

The development of hypertrophic scar after burn injury or surgery can result in significant cosmetic and functional disability to patients and surgeons. There are several studies to prove the effectiveness of different modalities in the treatment of hypertrophic scar. However, there is still the lack of a standardized, world-wide accepted and user-friendly tools for clinical use. Such an assessment tool must be able to quantify the longitudinal change of morphology and other properties of scar tissue, can reliably predict the progress and change of scar, and preferably with diagnostic value.

Usually, a descriptive scar index of the appearance including vascularity, texture, thickness above skin, and even pigmentation is adopted. They are not equally weighted and inter-examiner variability are very high.

In this thesis, two commercially available equipments were used. As there is no commonly accepted, objective assessment tool that can provide a scientifically documented reliable, external measurable criterion for comparison, it is not possible to compare the validity and reliability of using the cutometer and ultrasound imaging to clinical grading. This thesis can only correlate the result of the measurement with the traditional clinical grading. The reliability of the ultrasonography and elastometry were found to present with acceptable result. Concerning the assessment of the thickness of hypertrophic scar, use of ultrasonography is recommended. The ultrasonograph gives a comprehensive picture of the comparative thickness of the scar tissue. In the

assessment of the visco-elasticity of the scar, elastometer (cutometer SEM 757) is recommended. As R0 (the total deformation of skin in response to pressure) is thickness dependent, it can be helpful for longitudinal comparison with the corresponding normal skin. R2 or R5 reflecting different elasticity of the hypertrophic scar. The author believes that elasticity is not a good predictive value for the maturation/ remodelling of scar tissue. Higher fluctuation of the two values only reveal the dynamic process of collagen synthesis and degradation of an active scar, while in more mature scar, the magnitude of the fluctuation reduced. R8 is a more reliable value illustrating the visco-elasticity of scar tissue. Hypertrophic scar is more rigid and the response to suction may not resemble the characteristic of normal skin. This value gives a better predictive value that correlates with the clinical grading. In addition, a value of more than 0.193 defines a firm, active scar that need close monitoring.

In addition, a "Visco-elasticity-Thickness" chart is recommended for recording the change of the scar as a function of time. The application is well illustrated with some cases in the thesis.

Future Recommendation

Hypertrophic scar is rigid, (the ultrasonograph illustrates the depth of the scar to the dermis) and resistant to stretch. Therefore, the diameter of the measurement probe may affect the result. In this study, a standard measurement probe with 2mm in diameter is used. Though the size of probe should not affect the measurement of normal skin, another study on the potential effect of different sized probe on the

elasticity / visco-elasticity of hypertrophic scar is recommended. This may give a more comprehensive picture of the mechanical properties of hypertrophic scar.

A larger scale study of mature scar regarding different body regions is recommended. It is because the nature of mature scar may never resemble normal skin completely. So, comparative data of mature scar are essential for reference. This can help in the clinical judgement on when to stop conservative treatment, or to add on adjunctive treatment modalities such as injection, surgery etc.

A study of hypertrophic scar on donor site of different body regions is also important. It would serve to correlate skin tension with the development of hypertrophic scar. It is believed that skin tension of different body parts are not alike. A study of this area give more information for the choice of donor tissue that may have less chance of developing hypertrophy.

The predictive value of elasticity/ visco-elasticity can be used to predict and monitor the patients who are prone to develop hypertrophic scar. If normal skin of candidates with hypertrophic scar on donor site tends to have different values of R2, R5, R6 and R8, this indicates a different content of collagen activities. It would be helpful to the clinicians in deciding the choice of treatment for the burn wound and the expected prognosis.

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NO_PT	NAME	OT_CODE	DOB	DOI	1st AX	POST_IN	CAUSE	SEX	AGE	NO_SIT	SITE	NATURE
1	SO NGO FUNG	PS318	12/12/92	2/20/95	5/18/95	3	SCALD	M	2	1	14	HS
1	SO NGO FUNG	PS318	12/12/92	2/20/95	5/8/95	3	SCALD	M	2	2	14	HS
1	SO NGO FUNG	PS318	12/12/92	2/20/95	5/8/95	3	SCALD	M	2	3	14	HS
2	TONG WING SIZE	PB612	9/19/89	1/15/94	1/5/95	12	FRICTIONAL	F	5	1	1	HS
2	TONG WING SIZE	PB612		1/15/94	1/5/95	12	FRICTIONAL	F	5	2	1	HS
3	TONG SIU KUEN	PB190	4/6/78	12/12/87	4/8/95	92	SCALD	F	16	1	7	HS
3	TONG SIU KUEN	PB190	4/6/78	12/12/87	4/8/95	92	SCALD	F	16	2	9	HS
3	TONG SIU KUEN	PB190	4/6/78	12/12/87	4/8/95	92	SCALD	F	16	3	8	HS
3	TONG SIU KUEN	PB190	4/6/78	12/12/87	4/8/95	92	SCALD	F	16	4	8	infected
4	WAN CHUI TING	PS268	8/19/92	9/12/93	12/31/94	15	SCALD	F	2	1	20	skin graft
4	WAN CHUI TING	PS268	8/19/92	9/12/93	12/31/94	15	SCALD	F	2	2	17	HS
4	WAN CHUI TING	PS268	8/19/92	9/12/93	12/31/94	15	SCALD	F	2	3	16	skin graft
4	WAN CHUI TING	PS268	8/19/92	9/12/93	12/31/94	15	SCALD	F	2	4	7	HS
5	CHAU CHUN CHOI	PB621	12/21/86	4/21/94	1/4/95	9	SCALD	M	7	1	7	skin graft
5	CHAU CHUN CHOI	PB621	12/21/86	4/21/94	1/4/95	9	SCALD	M	7	2	8	HS
5	CHAU CHUN CHOI	PB621	12/21/86	4/21/94	1/4/95	9	SCALD	M	7	3	5	HS
5	CHAU CHUN CHOI	PB621	12/21/86	4/21/94	1/4/95	9	SCALD	M	7	4	5	HS
5	CHAU CHUN CHOI	PB621	12/21/86	4/21/94	1/4/95	9	SCALD	M	7	5	5	donor
6	CHEUNG SHUK YING	BS555	10/14/42	2/8/94	1/4/95	11	SCALD	F	52	1	5	HS
6	CHEUNG SHUK YING	BS555	10/14/42	2/8/94	1/4/95	11	SCALD	F	52	2	5	HS
6	CHEUNG SHUK YING	BS555	10/14/42	2/8/94	1/4/95	11	SCALD	F	52	3	5	donor
6	CHEUNG SHUK YING	BS555	10/14/42	2/8/94	1/4/95	11	SCALD	F	52	4	15	HS
6	CHEUNG SHUK YING	BS555	10/14/42	2/8/94	1/4/95	11	SCALD	F	52	5	15	HS
6	CHEUNG SHUK YING	BS555	10/14/42	2/8/94	1/4/95	11	SCALD	F	52	6	15	HS
7	CHEUNG TSZ KIN	PB620	10/27/87	9/20/94	1/5/95	3	WAX	M	7	1	5	HS
7	CHEUNG TSZ KIN	PB620	10/27/87	9/20/94	1/5/95	3	WAX	M	7	2	5	HS
7	CHEUNG TSZ KIN	PB620	10/27/87	9/20/94	1/5/95	3	WAX	M	7	2	5	HS
8	LAU SAU FAN	BO650	2/12/59	3/19/94	2/16/95	11	SCALD	F	35	1	7	HS
8	LAU SAU FAN	BO650	2/12/59	3/19/94	2/16/95	11	SCALD	F	35	2	8	HS
9	LEE SUK LING	BO682	5/7/52	2/26/95	4/29/95	2	SCALD	F	41	1	2	HS
9	LEE SUK LING	BO682	5/7/52	2/26/95	4/29/95	2	SCALD	F	41	2	3	HS
9	LEE SUK LING	BO682	5/7/52	2/26/95	4/29/95	2	SCALD	F	41	3	5	HS
9	LEE SUK LING	BO682	5/7/52	2/26/95	4/29/95	2	SCALD	F	41	4	5	HS
9	LEE SUK LING	BO682	5/7/52	2/26/95	4/29/95	2	SCALD	F	41	5	5	HS
9	LEE SUK LING	BO682	5/7/52	2/26/95	4/29/95	2	SCALD	F	41	6	3	HS

NO_PT	NAME	OT_CODE	DOB	DOI	1st AX	POST_IN	CAUSE	SEX	AGE	NO_SIT	SITE	NATURE
10	LAU YUK YEE	PB588	6/21/85	2/15/94	3/11/95	13	FLAME	F	9	1	5	skin graft
10	LAU YUK YEE	PB588	6/21/85	2/15/94	3/11/95	13	FLAME,BAN	F	9	2	5	HS
10	LAU YUK YEE	PB588	6/21/85	2/15/94	3/11/95	13	FLAME,D	F	9	3	5	donor
10	LAU YUK YEE	PB588	6/21/85	2/15/94	3/11/95	13	FLAME,D	F	9	4	5	donor
10	LAU YUK YEE	PB588	6/21/85	2/15/94	3/11/95	13	FLAME,INU	F	9	5	5	injected
11	YAU YUK TIN	BO664	12/26/59	10/27/94	1/3/95	2	SCALD+INF	M	35	1	4	HS
11	YAU YUK TIN	BO664	12/26/59	10/29/94	1/3/95	2	SCALD+INF	M	35	2	2	HS
11	YAU YUK TIN	BO664	12/26/59	10/29/94	1/3/95	2	SCALD+INF	M	35	3	1	HS
11	YAU YUK TIN	BO664	12/26/59	10/29/94	1/3/95	2	SCALD+INF	M	35	4	3	HS
11	YAU YUK TIN	BO664	12/26/59	10/29/94	1/3/95	2	SCALD+INF	M	35	5	1	HS
12	SUM WAN YEE	PB626	8/18/93	1/2/94	1/4/95	12	SCALD	F	0	1	1	HS
12	SUM WAN YEE	PB626	8/18/93	1/2/94	1/4/95	12	SCALD	F	0	2	1	HS
12	SUM WAN YEE	PB626	8/18/93	1/2/94	1/4/95	12	SCALD	F	0	3	14	injected
12	SUM WAN YEE	PB626	8/18/93	1/2/94	1/4/95	12	SCALD	F	0	4	5	HS
13	POON FUNG LIN	BO684	8/31/54	4/2/95	6/15/95	2	SCALD	F	41	1	5	HS
13	POON FUNG LIN	BO684	8/31/54	4/2/95	6/15/95	2	SCALD	F	41	2	3	HS
13	POON FUNG LIN	BO684	8/31/54	4/2/95	6/15/95	2	SCALD	F	41	3	2	HS
14	TSE SIU CHUNG	BS570	1/3/82	7/20/94	3/11/95	8	SCALD+INU	M	12	1	12	injected
14	TSE SIU CHUNG	BS570	1/3/82	7/20/94	3/11/95	8	SCALD	M	12	2	5	skin graft
14	TSE SIU CHUNG	BS570	1/3/82	7/20/94	3/11/95	8	SCALD	M	12	3	5	skin graft
14	TSE SIU CHUNG	BS570	1/3/82	7/20/94	3/11/95	8	SCALD	M	12	4	5	donor
15	WONG SUN SIM	BO660	3/3/34	7/1/94	1/5/95	6	SCALD	F	60	1	5	skin graft
15	WONG SUN SIM	BO660	3/3/34	7/1/94	1/5/95	6	SCALD	F	60	2	3	HS
15	WONG SUN SIM	BO660	3/3/34	7/1/94	1/5/95	6	SCALD	F	60	3	5	donor
16	YUNG KWOK LEUNG	PB564	12/20/83	9/1/90	8/19/95	59	WAX-1M-OP	M	11	1	6	HS
16	YUNG KWOK LEUNG	PB564	12/20/83	9/1/90	8/19/95	59	WAX-1M-OP	M	11	2	5	donor
16	YUNG KWOK LEUNG	PB564	12/20/83	9/1/90	8/19/95	59	WAX-1M-OP	M	11	3	4	skin graft
16	YUNG KWOK LEUNG	PB564	12/20/83	9/1/90	8/19/95	59	WAX-1M-OP	M	11	4	3	HS
16	YUNG KWOK LEUNG	PB564	12/20/83	9/1/90	8/19/95	59	WAX-1M-OP	M	11	5	2	HS
17	TAM SHIT YIN	BO634	12/30/44	1/5/93	1/28/95	24	COLD	M	50	1	10	skin graft
17	TAM SHIT YIN	BO634	12/30/44	1/5/93	1/28/95	24	COLD	M	50	2	10	HS
17	TAM SHIT YIN	BO634	12/30/44	1/5/93	1/28/95	24	COLD	M	50	3	10	skin graft
17	TAM SHIT YIN	BO634	12/30/44	1/5/93	1/28/95	24	COLD	M	50	4	10	HS

NO_PT	NAME	OT_CODE	DOB	DOI	1st AX	POST_IN	CAUSE	SEX	AGE	NO_SIT	SITE	NATURE
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD	F	4	1	5	HS
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD	F	4	2	5	HS
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD	F	4	3	5	HS
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD	F	4	4	5	HS
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD	F	4	5	3	HS
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD	F	4	6	3	HS
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD	F	4	7	2	HS
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD	F	4	8	1	skin graft
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD	F	4	9	2	HS
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD	F	4	10	6	HS
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD,MSG	F	4	11	5	skin graft
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD,D1	F	4	12	5	donor
18	KONG CHOR LING	PB581	6/14/89	1/19/94	1/5/95	12	SCALD,D2	F	4	13	5	injected

no. of pt	name	no. of site	site	nature	description
1	So N F	1	14		R back
		2	14		M back
		3	14		L back
2	Tong W S	1	1		L ankle front
		2	1		L ankle lateral
3	Tong S K	1	7		R arm
		2	9		R cutibal fossa
		3	8		R forearm
		4	8	injected	R forearm
4	Wan C T	1	20	SSG	R forehead
		2	17		R neck
		3	16	SSG	R chest
		4	7	SSG	R arm
5	Chau C C	1	7		R arm
		2	8		R forearm
		3	5		L thigh medial
		4	5		L thigh lateral
		5	5	D	R thigh
6	Cheung S Y	1	5		L thigh medial
		2	5		L thigh lateral
		3	5	D	R thigh posterior
		4	15		Abdomen 1
		5	15		Abdomen 2
		6	15		Abdomen 3
7	Cheung T K	1	5		L thigh proximal
		2	5		L thigh middle
		3	5		L thigh distal
8	Lau S F	1	7		L arm
		2	8		L forearm
9	Lee S L	1	2		R leg
		2	3		R knee
		3	5		R thigh, medial
		4	5		R thigh, proximal
		5	5		L thigh
		6	3		L knee
10	Lau Y Y	1	5	SSG	R thigh
		2	5		R thigh
		3	5	D	L thigh,
		4	5	D	L thigh, above knee
		5	5	injected	R thigh, lateral, near HS2
11	Yau Y T	1	4		L medial popliteal fossa
		2	2		L calf
		3	1		L, below med. malleolus
		4	3		R fibula head
		5	1		R, lat to lat. malleolus

12	Sum W Y	1	1		L med. malleolus
		2	1		R ankle, TA
		3	14	injected	R back
		4	5		L posterior thigh
13	Poon F L	1	5		L thigh
		2	3		R knee, medial
		3	2		R leg
14	Tse S C	1	12	injected	L groin
		2	5	SSG	L thigh
		3	5	SSG	R thigh
		4	5	D	L thigh
15	Wong S S	1	5		R thigh
		2	5	SSG	L thigh
		3	5	D	R knee
16	Yung K L	1	6		L hip, surgical scar
		2	5	D	L thigh, 5/52 post-op
		3	4	SSG	L popliteal, lateral
		4	3	SSG	L knee lateral
		5	2	chronic	L calf
17	Tam S Y	1	10	SSG	R wrist dorsum
		2	10		R wrist ventral
		3	10	SSG	L wrist dorsum
		4	10		L wrist ventral
18	Kong C L	1	5		L thigh
		2	5		L thigh
		3	5		L thigh
		4	5		L thigh
		5	3		L knee lateral
		6	3		L knee medial
		7	2		L leg lateral, above narrowest
		8	1	SSG	L ankle
		9	2		L leg lateral, mid-calf
		10	6		L hip
		11	5	SSG	L thigh
		12	5	D	R thigh, middle
		13	5	D,inj	R thigh, medial

NO	PT	NAME	CODE	DO_INJ	DO_AX	POST_INJ	CAUSE	DEG	SEX	AGE	NO	SITE	SITE	NATURE	G_1	G_2	G_3	G_4	G_5	G_6	G_7	G_8	G_9	G_10	G_11	G_12	G_13	G_14	G_15
1		SNF	PS318	2/20/95	5/18/95	3	SCALD	2	1	2.02	1	14	1	1	2	2	2	2	2	2	2	3	3		3	2	2	2	
1		SNF	PS318	2/20/95	5/8/95	3	SCALD	2	1	2.02	2	14	1	1	1	2	2	2	2	1	1	1			0	0	0		
1		SNG	PS318	2/20/95	5/8/95	3	SCALD	2	1	2.02	3	14	1	1	2	2	2	2	3	2	2	3	2		2	2	1	1	
2		TWS	PB612	1/15/94	1/5/95	12	FRICITIONAL	NA	2	5.00	1	1	1	1	3	2	2	2	2	2	2	3	1		1	1			
2		TWS	PB612	1/15/94	1/5/95	12	FRICITIONAL	NA	2	5.00	2	1	1	1	2	2	2	1	1	1	1	1		1	1	1			
3		TSK	PB190	12/12/87	4/8/95	92	SCALD	NA	2	16.00	1	7	1	1	3		3		3	3	2	2	2		2		2		
3		TSK	PB190	12/12/87	4/8/95	92	SCALD	NA	2	16.00	2	9	1	1	2		2		2	2	2	2	2		2		2		
3		TSK	PB190	12/12/87	4/8/95	92	SCALD	NA	2	16.00	3	8	1	1	3		3		2	2	2	2	2		2		1		
3		TSK	PB190	12/12/87	4/8/95	92	SCALD	NA	2	16.00	4	8	4	4					2	1	1	1	1		1		1		
4		WCT	PS268	9/12/93	12/31/94	15	SCALD	2	2	2.04	1	20	2	2	1					1			1						
4		WCT	PS268	9/12/93	12/31/94	15	SCALD	2	2	2.04	2	17	1	1	2					2				1					
4		WCT	PS268	9/12/93	12/31/94	15	SCALD	2	2	2.04	3	16	2	2	1					1					1				
4		WCT	PS268	9/12/93	12/31/94	15	SCALD	2	2	2.04	4	7	1	1	2					1									
5		CCC	PB621	4/21/94	1/4/95	9	SCALD	NA	1	7.04	1	7	2	2	3	2	2	2	2		2	2	2		2		1	1	
5		CCC	PB621	4/21/94	1/4/95	9	SCALD	NA	1	7.04	2	8	1	1	2	3	3	3	3		3	3	2		2		2	2	
5		CCC	PB621	4/21/94	1/4/95	9	SCALD	NA	1	7.04	3	5	1	1	4	4	3	2	2		2	2	2		2		2	2	
5		CCC	PB621	4/21/94	1/4/95	9	SCALD	NA	1	7.04	4	5	1	1	4	4	3	2	2		2	2	2		2		2	2	
5		CCC	PB621	4/21/94	1/4/95	9	SCALD	NA	1	7.04	5	5	3	3	1	1	1	1	1		1	1	1		1		1	1	
6		CSY	BS555	2/8/94	1/4/95	11	SCALD	2	2	52.00	1	5	1	1	3	3			2	2	3		3	2	2	2	2		
6		CSY	BS555	2/8/94	1/4/95	11	SCALD	2	2	52.00	2	5	1	1	3	3			3	3	2		3	2	2	2	2		
6		CSY	BS555	2/8/94	1/4/95	11	SCALD	2	2	52.00	3	5	3	3	3	3			3	3	3		3	3	2	2	2		
6		CSY	BS555	2/8/94	1/4/95	11	SCALD	2	2	52.00	4	15	1	1	3	3			3	3	3		3	3	3	3	3		
6		CSY	BS555	2/8/94	1/4/95	11	SCALD	2	2	52.00	5	15	1	1	3	3			3	3	3		3	3	3	3	3		
6		CSY	BS555	2/8/94	1/4/95	11	SCALD	2	2	52.00	6	15	1	1	3	3			3	3	3		3	3	3	3	3		
7		CSY	PB620	9/20/94	1/5/95	3	WAX	2	1	7.03	1	5	1	1	3				3	2		3	3	2	2	2	2		
7		CTK	PB620	9/20/94	1/5/95	3	WAX	2	1	7.03	2	5	1	1	3				3	2		3	3	2	2	2	2		
7		CTK	PB620	9/20/94	1/5/95	3	WAX	2	1	7.03	2	5	1	1	3				3	2		3	3	2	2	2	2		
8		LSF	BO650	3/19/94	2/16/95	11	SCALD	4	2	35.00	1	7	1	1	2	2	2			2	2		2	2	2	2			
8		LSF	BO650	3/19/94	2/16/95	11	SCALD	4	2	35.00	2	8	1	1	2	2	2			2	2		2	2	2	2			
9		LSL	BO682	2/26/95	4/29/95	2	SCALD	2	2	41.00	1	2	1	1	3	3	3	2	2	2	2		1	1	1	1			
9		LSL	BO682	2/26/95	4/29/95	2	SCALD	2	2	41.00	2	3	1	1	2	2	2	2	2	2	2		2	2	2	2			
9		LSL	BO682	2/26/95	4/29/95	2	SCALD	2	2	41.00	3	5	1	1	2	2	2	2	2	2	2		1	1	1	1			
9		LSL	BO682	2/26/95	4/29/95	2	SCALD	2	2	41.00	4	5	1	1	2	2	2	2	1	1	1		1	1	1	1			
9		LSL	BO682	2/26/95	4/29/95	2	SCALD	2	2	41.00	5	5	1	1	2	2	2	2	1	1	1		0	0	0	0			
9		LSL	BO682	2/26/95	4/29/95	2	SCALD	2	2	41.00	6	3	3	2	2			2	2	2	2	2		1	1	1	1		
10		LYY	PB588	2/15/94	3/11/95	13	FLAME	2	2	9.00	1	5	5	2	3					2	2	2	2		2	2	2		
10		LYY	PB588	2/15/94	3/11/95	13	FLAME,BAN	2	2	9.00	2	5	5	1	2					2	2	2	2		2	2	2		
10		LYY	PB588	2/15/94	3/11/95	13	FLAME,D	2	2	9.00	3	5	5	3	1					1	1	1	1		1	1	1		
10		LYY	PB588	2/15/94	3/11/95	13	FLAME,D	2	2	9.00	4	5	5	3						3	2	2	2		2	2	2		
10		LYY	PB588	2/15/94	3/11/95	13	FLAME,INU	2	2	9.00	5	5	4	4							1			1	1	1	1		

NO_PT	NAME	CODE	DO_INJ	DO_AX	POST_IN	CAUSE	EGR	SEX	AGE	NO_SITE	SITE	NATURE	G_1	G_2	G_3	G_4	G_5	G_6	G_7	G_8	G_9	G_10	G_11	G_12	G_13	G_14	G_15
11	YYT	BO664	10/27/94	1/3/95	2	SCALD+INF	2	1	35.00	1	4	1	3	3	3	3	3	2	2	2	2	2	2	2	1	1	1
11	YYT	BO664	10/29/94	1/3/95	2	SCALD+INF	2	1	35.00	2	2	1	3	3	2	2	2	2	2	2	2	2	2	2	2	1	1
11	YYT	BO664	10/29/94	1/3/95	2	SCALD+INF	2	1	35.00	3	1	1	3	3	2	3	3	3	3	2	2	2	3	2	2	2	2
11	YYT	BO664	10/29/94	1/3/95	2	SCALD+INF	2	1	35.00	4	3	1	4	4	3	2	2	2	2	2	2	2	2	2	2	2	2
11	YYT	BO664	10/29/94	1/3/95	2	SCALD+INF	2	1	35.00	5	1	1	1	3	3	3	3	3	3	3	2	2	2	2	2	2	2
12	SWY	PB626	1/2/94	1/4/95	12	SCALD	NA	2	.05	1	1	1	3	2	2		2	2	2	2	2	2	2	2	2	2	2
12	SWY	PB626	1/2/94	1/4/95	12	SCALD	NA	2	.05	2	1	1	3	2	2		2	2	2	2	2	2	2	2	2	2	2
12	SWY	PB626	1/2/94	1/4/95	12	SCALD	NA	2	.05	3	14	4	3	3	3		3	3	3	2	2	2	2	2	2	2	1
12	SWY	PB626	1/2/94	1/4/95	12	SCALD	NA	2	.05	4	5	1	2	2	2		2	2	2	3	2	2	2	2	2	2	2
13	PFL	BO684	4/2/95	6/15/95	2	SCALD	2	2	41.00	1	5	1	2	2	3	3	3	3	3	2	2	2	2	2	2	2	2
13	PFL	BO684	4/2/95	6/15/95	2	SCALD	2	2	41.00	2	3	1	2	2	2	3	3	3	3	2	2	2	2	2	2	2	3
13	PFL	BO684	4/2/95	6/15/95	2	SCALD	2	2	41.00	3	2	1	2	2	2	2	2	2	2	1	1	1	0	0	0	1	1
14	TSC	BS570	7/20/94	3/11/95	8	SCALD+INJ	2	1	12.02	1	12	4	3	3	3	2	2	2	2	2	2	1	1	1	1	1	1
14	TSC	BS570	7/20/94	3/11/95	8	SCALD	2	1	12.02	2	5	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
14	TSC	BS570	7/20/94	3/11/95	8	SCALD	2	1	12.02	3	5	2	1	1	1	1	1	1	1	1	1	1	1	0	0	0	0
14	TSC	BS570	7/20/94	3/11/95	8	SCALD	2	1	12.02	4	5	3	1	1	1	1	1	1	1	1	1	1	1	0	0	0	0
15	WSS	BO660	7/11/94	1/5/95	6	SCALD	2	2	60.00	1	5	2	2	2	2	1		1	1								
15	WSS	BO660	7/11/94	1/5/95	6	SCALD	1	2	60.00	2	3	1	2	2	2	1		1	1								
15	WSS	BO660	7/11/94	1/5/95	6	SCALD	2	2	60.00	3	5	3	1	1	1	0		0	0								
16	YKL	PB564	9/11/90	8/19/95	59	WAX-1M-OP	2	1	11.08	1	6	1	1	2	2	2	3	2	2	2	2	2	2	2	2	2	2
16	YKL	PB564	9/11/90	8/19/95	59	WAX-1M-OP	2	1	11.08	2	5	3	2	2	2	2	2	2	2	2	2	2	2	1	1	1	1
16	YKL	PB564	9/11/90	8/19/95	59	WAX-1M-OP	2	1	11.08	3	4	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
16	YKL	PB564	9/11/90	8/19/95	59	WAX-1M-OP	2	1	11.08	4	3	1	2	3	3	2	2	2	2	2	2	2	2	2	2	2	2
16	YKL	PB564	9/11/90	8/19/95	59	WAX-1M-OP	2	1	11.08	5	2	1	3	3	3	3	2	3	3	3	3	3	3	3	3	3	3
17	TSY	BO634	1/5/93	1/28/95	24	COLD	NA	1	50.00	1	10	2	2		2	2		2									1
17	TSY	BO634	1/5/93	1/28/95	24	COLD	NA	1	50.00	2	10	1	2		2	2		1									1
17	TSY	BO634	1/5/93	1/28/95	24	COLD	NA	1	50.00	3	10	2	2		2	2		1									1
17	TSY	BO634	1/5/93	1/28/95	24	COLD	NA	1	50.00	4	10	1	1		1	1		1									1
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	1	5	1	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	2	5	1	3	3	3	3	3	3	3	2	2	2	2	2	2	2	2
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	3	5	1	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	4	5	1	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	5	3	1	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	6	3	1	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	7	2	1	3	3	3	3	3	3	3	2	2	2	2	2	2	2	2
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	8	1	2	4	4	4	4	4	4	4	2	2	2	2	2	2	2	2
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	9	2	1	3	3	3	3	3	3	3	2	2	2	2	2	2	2	2
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	10	6	1	3		3	3	3	3	3	2	2	2	2	2	2	2	2
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	11	5	2	1			1	1	1	1	1	1	1	1	1	1	1	1
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	12	5	3	3			4	4	4	4	3	3	3	3	3	3	3	3
18	KCL	PB581	1/19/94	1/5/95	12	SCALD	3	2	4.07	13	5	4	4		4	4	4	4	4	3	3	3	3	3	3	3	3

Measurement of the Visco-elastic properties

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 621	HS1 R ARM, SSG, ACTIVE	1/4/95	9.11	HS1 R ARM	0.300	0.833	0.600	0.200	0.117	CCC1
PB 621	NS2 L ARM, VS CCC1	1/4/95	9.12	NS2 L ARM	0.000	0.000	0.000	0.000	0.000	CCC2-NS
PB 621	HS3 R FOREARM, ACTIVE, HYP	1/4/95	9.14	HS3 R FORE	0.210	0.762	0.800	0.400	0.186	CCC3
PB 621	NS L FOREARM VS CCC3	1/4/95	9.15	NS4 L FORE	0.290	0.897	1.000	0.261	0.153	CCC4-NS
PB 621	HS5 L KNEE, MEDIAL, FIRM SC	1/4/95	9.16	HS5 L KNEE	0.130	0.846	0.700	0.300	0.226	CCC5
PB 621	HS6 L KNEE LATERAL, ACTIVE	1/4/95	9.18	HS6 L KNEE	0.100	0.600	0.800	1.000	0.465	CCC6
PB 621	NS R KNEE, VS CCC5 & CCC6	1/4/95	9.19	NS7	0.340	0.971	1.032	0.097	0.107	CCC7-NS
PB 621	HS8 R THIGH DONAR, MILD HS	1/4/95	9.20	HS8 R THI	0.180	0.833	0.714	0.286	0.192	CCC8-D
PB 621	NS9 L THIGH VS CCC8	1/4/95	9.21	NS9 L THI	0.380	0.974	1.029	0.118	0.094	CCC9-NS
PB 621	HS1 R ARM SSG, 1YR POST IN	3/11/95	10.22	HS1 R ARM	0.280	0.857	0.696	0.217	0.130	CCC3-1
PB 621	NS L THIGH	3/11/95	10.38	NS L THI	0.330	0.970	1.034	0.138	0.084	CCC3-11N
PB 621	HS2 R ELBOW	3/11/95	10.24	HS2 R ELB	0.290	0.724	0.682	0.318	0.141	CCC3-2
PB 621	HS3 L THIGH MEDIAL ASPECT	3/11/95	10.26	HS3 L THI	0.110	0.909	0.875	0.375	0.183	CCC3-3
PB 621	HS4 L THIGH MEDIAL ASPECT	3/11/95	10.28	HS4 L THI	0.120	0.833	0.875	0.500	0.341	CCC3-4
PB 621	HS5 R THIGH, DONAR	3/11/95	10.29	HS5 R THI	0.230	0.913	0.842	0.211	0.119	CCC3-5
PB 621	NS L ARM VS CCC3-1	3/11/95	10.31	NS L ARM	0.420	0.952	1.027	0.135	0.074	CCC3-6NS
PB 621	NS L ELBOW VS CCC3-2	3/11/95	10.33	NS L ELBOW	0.380	0.947	1.000	0.152	0.102	CCC3-7NS
PB 621	NS L THIGH, VS CCC3-3	3/11/95	10.35	NS L THI	0.350	0.943	0.968	0.129	0.103	CCC3-8NS
PB 621	NS R THIGH VS CCC3-4	3/11/95	10.36	NS R THI	0.530	0.981	1.000	0.082	0.082	CCC3-9NS
PB 612	R ARM, HS1, SSG	950413	16.16	R ARM, HS1	0.320	0.938	1.000	0.280	0.109	CCC4-1
PB 612	R FOREARM, HS2, ACTIVE, RAI	950413	16.18	R FARM, HS2	0.330	0.848	0.800	0.320	0.199	CCC4-2
PB 612	R THIGH, ACTIVE HS3	950413	16.19	R THI, HS3	0.160	0.813	0.909	0.455	0.289	CCC4-3
PB 612	L THIGH, HS4, ACTIVE	950413	16.20	L THI, HS4	0.140	0.857	0.778	0.556	0.294	CCC4-4
PB 612	R THIGH, DONAR	950413	16.22	R THI, D	0.300	0.900	0.917	0.250	0.110	CCC4-D
PB 621	HS1 R ARM, SSG	950518	15.46	HS1 R ARM	0.280	0.893	0.864	0.273	0.165	CCC5-1
PB 621	HS3 R FOREARM	950518	15.49	HS3 R FORA	0.280	0.750	0.609	0.217	0.114	CCC5-3
PB 621	HS3 R FOREARM	950518	15.50	HS3 R FORA	0.340	0.853	0.821	0.214	0.137	CCC5-3
PB 621	HS4 L KNEE MEDIAL ASPECT	950518	15.51	HS4 L KNEE	0.130	0.769	0.727	0.182	0.121	CCC5-4
PB 621	HS5 L THIGH LATERAL	950518	15.53	HS5 L THI	0.150	0.867	0.800	0.500	0.273	CCC5-5
PB 621	HS5 R THIGH, DONAR	950518	15.54	HS6 R THI	0.450	0.978	1.132	0.184	0.145	CCC5-6
PB 621	NS R THIGH MEDIAL TO DONA	950518	15.55	NS R THI	0.370	0.946	1.030	0.121	0.095	CCC5-NS
PB 621	HS1 SSG, R ARM	950629	15.53	HS1 R ARM	0.370	0.919	0.968	0.194	0.112	CCC6-1
PB 621	HS1 R ARM SSG, 2ND MX	950629	15.54	HS1 R ARM	0.360	0.861	0.862	0.241	0.116	CCC6-1B
PB 621	NS1 L ARM	950629	15.59	NS1 L ARM	0.600	0.983	1.019	0.111	0.082	CCC6-1NS
PB 621	HS2 R FOREARM,	950629	15.55	HS2 R FORE	0.330	0.788	0.615	0.269	0.157	CCC6-2
PB 621	NS2 L FOREARM	950629	16.00	NS2 L FORE	0.580	0.983	1.060	0.160	0.107	CCC6-2NS
PB 621	HS3 L THIGH, MEDIAL	950629	15.56	HS3 L THI	0.140	0.714	0.636	0.273	0.153	CCC6-3
PB 621	NS3 R THIGH, VS HS3 & HS4	950629	16.01	NS3 R THI	0.440	0.977	1.026	0.128	0.071	CCC6-3NS
PB 621	HS4 L THIGH, LATERAL	950629	15.57	HS4 L THI	0.100	0.800	0.571	0.429	0.175	CCC6-4
PB 621	HS5 R THIGH, DONOR	950629	15.58	HS5 R THI	0.170	0.941	1.000	0.308	0.222	CCC6-5
PB 621	NS5 L THIGH, PROXIMAL	950629	16.02	NS5 L THI	0.460	0.957	1.025	0.150	0.103	CCC6-5NS
PB 621	HS1 R FOREARM,	950727	15.49	HS1 R FORA	0.270	0.926	0.727	0.227	0.138	CCC7-1
PB 621	HS2 L ARM	950727	15.50	HS2 L ARM	0.150	0.800	1.333	0.667	0.166	CCC7-2
PB 621	HS3 L THIGH ABOVE KNEE	950727	15.51	HS3 L THI	0.200	0.550	0.636	0.818	0.468	CCC7-3
PB 621	HS4 R THIGH, DONOR	950727	15.53	HS4 R THI	0.210	0.905	0.778	0.167	0.102	CCC7-4D
PB 621	HS1 R ARM, SSG	950907	16.11	HS1 R ARM	0.350	0.857	0.800	0.167	0.111	CCC8-1
PB 621	HS2 R FOREARM	950907	16.11	HS2 R FORM	0.190	0.789	0.600	0.267	0.131	CCC8-2
PB 621	HS3 R THIGH, MEDIAL	950907	16.13	HS3 L THI	0.210	1.000	1.176	0.235	0.110	CCC8-3
PB 621	HS4 L THIGH, LATERAL	950907	16.13	HS4 L THI	0.220	0.773	0.529	0.294	0.140	CCC8-4
PB 621	HS5 R THIGH, DONOR	950907	16.14	HS5 R THI	0.380	0.737	0.618	0.118	0.082	CCC8-5
PB 621	HS1 R ARM, SSG	950930	12.00	HS1 R ARM	0.240	0.917	0.842	0.263	0.182	CCC9-1
PB 621	NS1 L ARM	950930	12.04	NS1 L ARM	0.550	0.964	0.942	0.058	0.042	CCC9-1NS
PB 621	HS2 R FOREARM	950930	12.01	HS2 R FORA	0.330	0.848	0.654	0.269	0.139	CCC9-2
PB 621	NS2 L FOREARM	950930	12.04	NS2 L FORA	0.420	0.952	1.027	0.135	0.106	CCC9-2NS
PB 621	HS3 L THIGH, MEDIAL	950930	12.01	HS3 L THI	0.180	0.944	0.857	0.286	0.114	CCC9-3
PB 621	NS3 R THIGH, NEAR KNEE	950930	12.05	NS3 R THI	0.330	0.970	1.034	0.138	0.092	CCC9-3NS
PB 621	HS4 L THIGH, LATERAL	950930	12.02	HS4 L THI	0.160	0.688	0.455	0.455	0.205	CCC9-4
PB 621	HS5 R THIGH, DONOR	950930	12.03	HS5 R THI	0.250	0.960	0.950	0.250	0.130	CCC9-5
PB 621	NS5 L THIGH	950930	12.06	NS5 L THI	0.340	0.941	1.000	0.172	0.115	CCC9-5NS
PB 621	HS1 R ARM, SSG	951111	11.45	HS1 R ARM	0.430	0.744	0.658	0.132	0.086	CCC10-1
PB 621	HS2 R FOREARM	951111	11.46	HS2 R FORM	0.550	0.600	0.383	0.170	0.128	CCC10-2
PB 621	HS3 L THIGH, MEDIAL	951111	11.47	HS3 L THI	0.310	1.000	0.808	0.192	0.088	CCC10-3
PB 621	HS4 L THIGH, LATERAL	951111	11.48	HS4 L THI	0.240	0.792	0.650	0.200	0.105	CCC10-4
PB 621	HS5 R THIGH, DONOR	951111	11.48	HS5 R THI	0.230	0.826	0.556	0.278	0.205	CCC10-5
PB 621	HS1 R ARM, SSG	951223	10.54	HS1 R ARM	0.390	0.846	0.676	0.147	0.087	CCC11-1
PB 621	HS1 R ARM, SSG, 2ND MX	951223	10.55	HS1 R ARM	0.400	0.950	0.853	0.176	0.102	CCC11-1B
PB 621	HS2 R FOREARM,	951223	10.57	HS2 R FORM	0.230	0.826	0.556	0.278	0.113	CCC11-2
PB 621	HS2 R FOREARM, 2ND MX	951223	10.57	HS2 R FORM	0.240	0.667	0.474	0.263	0.178	CCC11-2B
PB 621	HS3 R THIGH, MEDIAL	951223	10.58	HS3 L THI	0.200	0.800	0.667	0.333	0.190	CCC11-3
PB 621	HS3 R THIGH, MEDIAL 2ND MX	951223	10.59	HS3 L THI	0.190	0.842	0.688	0.187	0.102	CCC11-3B
PB 621	HS4 L THIGH, LATERAL	951223	11.00	HS4 L THI	0.260	0.846	0.500	0.300	0.139	CCC11-4
PB 621	HS4 L THIGH, LATERAL	951223	11.00	HS4 L THI	0.220	0.727	0.437	0.375	0.144	CCC11-4B
PB 621	HS5 R THIGH, DONOR 2ND MX	951223	11.02	HS5 R THI	0.280	0.857	0.826	0.217	0.128	CCC11-5B

PB 621	HS5 R THIGH, DONOR, 3RD MX	951223	11.03	HS5 R THI	0.190	0.947	0.733	0.267	0.157	CCC11-5C
PB 621	NS1 L ARM	951223	11.04	NS1 L ARM	0.420	1.000	1.056	0.167	0.101	CCC11-N1
PB 621	NS2 L FOREARM	951223	11.05	NS2 L FORM	0.420	0.976	0.974	0.105	0.060	CCC11-N2
PB 621	NS3 R THIGH, MEDIAL	951223	11.05	NS3 R THI	0.410	0.951	1.029	0.171	0.114	CCC11-N3
PB 621	NS4 R THIGH, LATERAL	951223	11.07	NS4 R THI	0.300	0.967	1.038	0.154	0.088	CCC11-N4
PB 621	NS5 L THIGH	951223	11.07	NS5 L THI	0.460	0.978	1.024	0.095	0.084	CCC11-N5
PB 621	HS1 R ARM,SSG	960203	11.52	HS1 R ARM	0.450	0.778	0.703	0.216	0.123	CCC12-1
PB 621	HS2 R FOREARM	960203	11.53	HS2 R FORM	0.280	0.607	0.550	0.400	0.202	CCC12-2
PB 621	HS3 L THIGH,MEDIAL	960203	11.54	HS3 L THI	0.350	0.600	0.500	0.250	0.147	CCC12-3
PB 621	HS4 L THIGH,LATERAL	960203	11.54	HS4	0.350	0.571	0.429	0.250	0.108	CCC12-4
PB 621	HS5 R THIGH,DONORH	960203	11.55	HS5	0.280	0.786	0.739	0.217	0.098	CCC12-5
PB 621	NS1 L ARM	960203	11.56	NS1 R ARM	0.600	0.867	0.942	0.154	0.084	CCC12-N1
PB 621	NS2 L FOREARM	960203	11.56	NS2 R FORM	0.660	0.818	0.831	0.119	0.064	CCC12-N2
PB 621	NS3 R THIGH,DISTAL	960203	11.57	NS3 R THI	0.570	0.825	0.830	0.075	0.043	CCC12-N3
PB 621	NS4 L THIGH	960203	11.58	NS4 L THI	0.610	0.820	0.870	0.130	0.080	CCC12-N4
PB 621	HS1 R ARM, SSG	960302	12.01	HS1 R ARM	0.470	0.872	0.707	0.146	0.070	CCC13-1
PB 621	HS2 R FOREARM	960302	12.04	HS2 R FORA	0.550	0.545	0.348	0.196	0.124	CCC13-2
PB 621	HS3 L THIGH,MEDIAL	960302	12.05	HS3 L THI	0.270	1.000	1.000	0.286	0.132	CCC13-3
PB 621	HS4 L THIGH,LATERAL	960302	12.05	HS4 L THI	0.380	0.421	0.258	0.226	0.119	CCC13-4
PB 621	D1 R THIGH, MATURE	960302	12.06	D1 R THI	0.410	1.000	1.206	0.206	0.133	CCC13-D
PB 621	NS1 L ARM	960302	12.07	NS1 L ARM	0.830	0.843	0.851	0.122	0.073	CCC13-N1
PB 621	NS2 L FOREARM	960302	12.07	NS2 L FORA	0.620	1.000	1.107	0.107	0.073	CCC13-N2
PB 621	NS3 R THIGH, DISTAL TO D1	960302	12.08	NS3 R THI	0.480	1.000	1.116	0.116	0.070	CCC13-N3
PB 621	HS1 R ARM, SSG	960420	11.22	HS1 L ARM	0.300	0.933	0.920	0.200	0.129	CCC14-1
PB 621	HS2 L ELBOW, MATURE	960420	11.23	HS2 L ELB	0.280	0.893	0.792	0.167	0.090	CCC14-2
PB 621	HS3 L KNEE, LATEREAL	960420	11.24	HS3 R KNEE	0.210	0.857	0.688	0.312	0.147	CCC14-2
PB 621	HS4 R THIGH, DONOR	960420	11.25	S4 R THIGH	0.360	0.861	0.806	0.161	0.131	CCC14-4
PB 621	NS1 L ARM	960420	11.26	NS1 L ARM	0.570	0.982	1.000	0.096	0.067	CCC14-N1
PB 621	NS2 L ELBOW	960420	11.27	NS2 L ELB	0.510	0.961	0.957	0.085	0.059	CCC14-N2
PB 621	NS3 R KNEE LATERAL	960420	11.28	NS3 R KNEE	0.340	0.971	1.000	0.097	0.064	CCC14-N3
PB 621	NS4 THIGH	960420	11.25	NS4 L THI	0.560	1.000	0.962	0.077	0.052	CCC14-N4
PB 621	HS1 R ARM	960601	11.02	HS1 R ARM	0.260	0.923	0.818	0.182	0.119	CCC16-1
PB 621	HS3 R FOREARM	960601	11.03	HS3 R FORM	0.210	0.857	0.706	0.235	0.152	CCC16-3
PB 621	HS4 L KNEE	960601	11.04	HS4 L KNEE	0.180	0.889	0.733	0.200	0.101	CCC16-4
PB 621	D1 R THIGH	960601	11.05	D1 R THI	0.260	0.885	0.773	0.182	0.106	CCC16-D1
PB 621	NS1 L ARM	960601	11.06	N1 L ARM	0.460	0.935	0.951	0.122	0.066	CCC16-N1
PB 621	NS3 L FOREARM	960601	11.07	N3 L FORM	0.440	0.955	1.026	0.128	0.081	CCC16-N3
PB 621	NS4 R KNEE	960601	11.08	N4 R KNEE	0.330	0.939	0.967	0.100	0.050	CCC16-N4
PB 621	NS5 L THIGH	960601	11.08	N5 L THI	0.410	0.951	1.000	0.108	0.072	CCC16-N5

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
BS 555	HS1 L THIGH MEDIAL ASPECT,	1/4/95	9.57	HS1 L THI	0.130	0.923	0.800	0.300	0.200	CSY1
BS 555	NS ABDOMEN VS CSY7,8 & 9	1/4/95	10.08	NS ABDOM	0.360	0.972	0.909	0.091	0.058	CSY10
BS 555	NS ABDOMEN VS CSY7,8 & 9	1/4/95	10.08	NS ABDOM	0.360	0.972	0.909	0.091	0.058	CSY10-NS
BS 555	HS2 L THIGH, LATERAL ASPEC	1/4/95	9.58	HS2 L THI	0.100	0.800	0.714	0.429	0.179	CSY2
BS 555	NS, R THIGH VS CSY1 & CSY2	1/4/95	9.59	NS R THI	0.570	0.982	1.020	0.118	0.098	CSY3
BS 555	NS R THIGH, VS CSY1 & CSY2	1/4/95	9.59	NS R THI	0.570	0.982	1.020	0.118	0.098	CSY3-NS
BS 555	HS4 R THIGH, DONAR SITE,	1/4/95	10.00	HS4 R THI	0.160	0.875	0.727	0.455	0.249	CSY4
BS 555	HS5 R THIGH, MATURE DONAR	1/4/95	10.01	HS5 R THI	0.410	0.951	0.946	0.108	0.074	CSY5
BS 555	NS L THIGH, POSTERIOR ASP	1/4/95	10.03	NS L THI	0.420	0.952	1.000	0.135	0.098	CSY6-NS
BS 555	HS7, ABDOMEN1	1/4/95	10.05	HS7 ABDOM	0.220	0.636	0.533	0.467	0.199	CSY7
BS 555	HS8, ABDOMEN 2	1/4/95	10.06	HS8 ABDOM	0.130	0.769	0.750	0.625	0.447	CSY8
BS 555	HS9, ABDOMEN 3	1/4/95	10.07	HS9 ABDOM	0.200	0.850	0.733	0.333	0.174	CSY9
BS 555	HS1 L THIGH, MEDIAL	950629	16.23	HS1 L THI	0.260	0.923	0.818	0.182	0.105	CSY3-1
BS 555	NS1 R THIGH VS HS1, HS2	950629	16.28	NS1 R THI	0.410	0.951	1.029	0.171	0.115	CSY3-1NS
BS 555	HS2 L THIGH, LATERAL	950629	16.24	HS2 L THI	0.190	0.895	0.857	0.357	0.223	CSY3-2
BS 555	HS3 R THIGH, POSTERIOR	950629	16.24	HS3 R THI	0.070	0.857	0.800	0.400	0.312	CSY3-3
BS 555	NS2 L THIGH POSTERIOR, VS	950629	16.29	NS2 L THI	0.500	0.960	1.045	0.136	0.082	CSY3-3NS
BS 555	HS4 ABDOMEN 1	950629	16.26	HS4 ABDOM	0.220	1.000	0.824	0.294	0.142	CSY3-4
BS 555	NS3 ABDOMEN	950629	16.30	NS3 ABDOM	0.520	0.942	0.933	0.156	0.112	CSY3-4NS
BS 555	HS5 ABDOMEN 2	950629	16.26	HS4 ABDOM	0.100	1.000	1.667	0.667	0.385	CSY3-5
BS 555	HS6 ABDOMEN3	950629	16.27	HS6 ABDOM	0.150	0.800	0.800	0.500	0.191	CSY3-6
BS 555	HS1 L THIGH	950727	17.03	HS1 L THI	0.390	0.538	0.429	0.114	0.049	CSY4-1
BS 555	HS1 L THIGH	950727	17.05	HS1 L THI	0.240	0.667	0.524	0.143	0.091	CSY4-1
BS 555	HS2 L THIGH JUST ABOVE KN	950727	17.04	HS2 L KNEE	0.190	0.842	0.667	0.267	0.178	CSY4-2
BS 555	HS3 R THIGH, POSTERIOR	950727	17.06	HS3 R THI	0.080	0.875	0.833	0.333	0.199	CSY4-3
BS 555	HS4 ABODMEN 1	950727	17.07	HS4 ABDO1	0.270	0.815	0.650	0.350	0.280	CSY4-4
BS 555	HS5 ABDOMEN 2	950727	17.08	HS4 ABDO1	0.130	0.615	0.600	0.300	0.246	CSY4-5
BS 555	HS6 ABDOMEN 3	950727	17.09	HS6 ABDO3	0.150	0.800	0.455	0.364	0.157	CSY4-6
PB 588	HS1 L THIGH 1	950916	11.13	HS1 L THI	0.290	0.621	0.542	0.208	0.137	CSY5-1
BS555	NS1 R THIGH, VS HS1, HS2,	950916	11.19	NS1 R THI	0.390	0.744	0.735	0.147	0.074	CSY5-1NS
BS555	HS2 L THIGH, DISTAL	950916	11.14	HS2 L THI2	0.240	0.833	0.650	0.200	0.112	CSY5-2
BS555	NS2 L THIGH POSTERIOR, VS	950916	11.20	NS2 L THI	0.370	1.000	0.818	0.121	0.098	CSY5-2NS
BS555	HS3 R THIGH, POSTERIOR, DO	950916	11.15	HS3 R THI	0.140	0.786	0.700	0.400	0.282	CSY5-3D
BS555	NS3 ABDOMEN	950916	11.18	NS3 ABD	0.520	0.942	0.938	0.083	0.051	CSY5-3NS
BS555	HS4 ABDOMEN 1	950916	11.15	HS4 ABD1	0.250	0.720	0.632	0.316	0.193	CSY5-4
BS555	HS5 ABDOMEN 2	950916	11.16	HS5 ABD2	0.200	1.000	1.250	0.250	0.173	CSY5-5
BS555	HS6 ABDOMEN 3	950916	11.17	HS6 ABD 3	0.120	0.917	0.889	0.333	0.180	CSY5-6
BS 555	HS1 L THIGH, MEDIAL	951014	11.07	HS1 L THI	0.390	0.744	0.562	0.219	0.128	CSY6-1
BS 555	HS2 L THIGH, LATERAL	951014	11.08	HS2 L THI	0.400	0.875	0.686	0.143	0.104	CSY6-2
BS 555	HS3 R THIGH, DONOR	951014	11.08	HS3 R THI	0.250	0.720	0.500	0.250	0.167	CSY6-3
BS 555	HS4 ABDOMEN 1	951014	11.09	HS4 ABD1	0.430	0.581	0.444	0.194	0.130	CSY6-4
BS 555	HS5 ABDOMEN 2	951014	11.10	HS5 ABD 2	0.350	1.000	1.129	0.129	0.078	CSY6-5
BS 555	HS6 ABDOMEN 3	951014	11.11	HS6 ABD 3	0.160	0.938	0.692	0.231	0.128	CSY6-6
BS 555	HS1 L THIGH, MEDIAL	951111	11.31	HS1 L THI	0.370	0.919	0.485	0.121	0.099	CSY8-1
BS 555	HS2 L THIGH, LATERAL	951111	11.32	HS2 L THI	0.430	0.907	0.737	0.132	0.068	CSY8-2
BS 555	HS3 R THIGH, POSTERIOR	951111	11.33	HS3 R THI	0.190	0.789	0.615	0.462	0.270	CSY8-3
BS 555	HS4 ABDOMEN 1, R SIDE	951111	11.34	HS4 ABDO-1	0.150	0.933	0.818	0.364	0.233	CSY8-4
BS 555	HS5 ABDOMEN 2, R SIDE	951111	11.35	HS5 ABD-2	0.220	1.000	1.294	0.294	0.174	CSY8-4
BS 555	HS5 ABDOMEN 2, L SIDE	951111	11.35	HS5 ABD-2	0.220	1.000	1.294	0.294	0.174	CSY8-5
BS 555	HS6 ABDOMEN 3, LOWEST	951111	11.35	HS6 ABD-3	0.290	1.000	0.952	0.381	0.171	CSY8-6
BS 555	HS1 L THIGH, MEDIAL	951209	12.09	HS1 L THI	0.250	1.000	1.190	0.190	0.144	CSY9-1
BS 555	HS2 L THIGH, LATERAL	951209	12.11	HS2 L THI	0.310	1.000	0.760	0.240	0.141	CSY9-2
BS 555	HS3 R THIGH, POSTERIOR, DO	951209	12.11	HS3 R THI	0.280	0.643	0.500	0.273	0.196	CSY9-3
BS 555	HS3 ABDOMEN1,	951209	12.12	HS4 ABD1	0.410	0.610	0.437	0.281	0.124	CSY9-4
BS 555	HS5 ABDOMEN 2	951209	12.13	HS5 ABD2	0.330	1.000	1.185	0.222	0.104	CSY9-5
BS 555	HS6 ABDOMEN3	951209	12.14	HS6 ABD3	0.120	0.750	0.667	0.333	0.251	CSY9-6
BS 555	HS1 L THIGH MEDIAL	960106	11.58	HS1 L THI	0.400	0.850	0.686	0.143	0.136	CSY10-1
BS 555	HS2 L THIGH, LATERAL	960106	11.59	HS2 L THI	0.390	0.769	0.647	0.147	0.119	CSY10-2
BS 555	HS4 R THIGH, POSTERIOR, DO	960106	12.00	HS3 R THI	0.150	0.867	0.727	0.364	0.183	CSY10-3D
BS 555	HS4 ABDOMEN 1	960106	12.01	HS4 ABD 1	0.240	0.917	0.737	0.263	0.190	CSY10-4
BS 555	HS5 ABDOMEN 2,	960106	12.02	HS5 ABD 2	0.140	0.786	0.700	0.400	0.221	CSY10-5
BS 555	HS6 ABDOMEN 3	960106	12.03	HS6 ABD 3	0.240	0.750	0.687	0.500	0.085	CSY10-6
BS 555	NS1 R THIGH	960106	12.04	NS1 R THI	0.420	0.929	0.919	0.135	0.101	CSY10-N1
BS 555	NS2 ABDOMEN	960106	12.05	NS2 ABD	0.610	0.934	0.929	0.089	0.059	CSY10-N2

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 581	HS1 L THIGH, ACTIVE, 1YR PO	1/5/95	14.44	HS1 L THI	0.080	0.750	0.667	0.333	0.181	KCL1
PB 581	HS2 L THIGH	1/5/95	14.45	HS2 L THI	0.050	0.800	1.000	0.667	0.301	KCL2
PB 581	HS3 L THIGH	1/5/95	14.46	HS3 L THI	0.070	0.857	0.800	0.400	0.280	KCL3
PB 581	HS4 L THIGH	1/5/95	14.47	HS4 L THI	0.080	0.750	0.500	0.333	0.168	KCL4
PB 581	HS5 L THIGH	1/5/95	14.48	HS5 L THI	0.080	0.750	0.833	0.333	0.196	KCL5
PB 581	HS6 L THIGH	1/5/95	14.49	HS6 L THI	0.050	1.000	1.000	0.667	0.532	KCL6
PB 581	HS7 L THIGH	1/5/95	14.50	HS7 L THI	0.040	1.000	1.000	0.333	0.128	KCL7
PB 581	HS8 L THIGH	1/5/95	14.52	HS8 L THI	0.070	0.857	1.000	0.750	0.345	KCL8
PB 581	HS9 L THIGH	1/5/95	14.53	HS9 L THI	0.040	0.750	1.000	0.333	0.092	KCL9
PB 581	HS10 L HIP	1/5/95	14.55	HS10 L HIP	0.110	0.909	0.667	0.222	0.128	KCL10
PB 581	HS11 L THIGH, MATURE SSG	1/5/95	14.57	HS11 L THI	0.190	0.947	1.000	0.187	0.115	KCL11-SG
PB 581	HS12 R THIGH, DONAR 1	1/5/95	14.59	HS12 R THI	0.070	0.857	1.000	0.750	0.465	KCL12
PB 581	HS13 R THIGH, DONAR 2, 1 YR	1/5/95	15.01	HS13 R THI	0.070	0.714	1.000	0.750	0.373	KCL13
PB 581	NS R THIGH, VS KCL 1,2,4 & 11	1/5/95	15.04	NS R THI	0.270	0.963	1.091	0.227	0.171	KCL14-NS
PB 581	NS R THIGH, VS KCL 5	1/5/95	15.05	NS R THI	0.500	0.980	1.022	0.111	0.076	KCL15-NS
PB 581	NS R KNEE VS KCL6	1/5/95	15.06	NS R KNEE	0.440	0.932	1.054	0.189	0.123	KCL16-NS
PB 581	NS R LEG, VS KCL9	1/5/95	15.07	NS R LEG	0.290	0.931	1.040	0.160	0.101	KCL17-NS
PB 581	NS R LEG, VS KCL7	1/5/95	15.07	NS R LEG	0.190	0.895	1.000	0.357	0.227	KCL18-NS
PB 581	NS R ANKLE VS KCL8	1/5/95	15.09	NS R ANK	0.000	0.000	0.000	0.000	0.000	KCL19-NS
PB 581	HS1 L HIP	2/22/96	14.08		0.190	0.842	0.688	0.187	0.214	KCL15-1
PB 581	HS6 L KNEE, ACTIVE	5/15/95	1.01	HS6 L KNEE	0.050	1.000	1.000	0.667	0.532	KCL6
PB 581	HS7 L LEG, ACTIVE, 1 YR POS	5/15/95	1.02	HS7 L LEG	0.040	1.000	1.000	0.333	0.128	KCL7
PB 581	HS8 L ANKLE, ACTIVE	5/15/95	1.02	HS8 L ANK	0.070	0.857	1.000	0.750	0.345	KCL8
PB 581	HS9 L LEG, ACTIVE	5/15/95	1.03	HS9 L LEG	0.040	0.750	1.000	0.333	0.092	KCL9
PB 581	L HIP HS, FIRM	950302	16.37	L HIP/HS	0.230	0.913	0.722	0.278	0.221	KCL3-10
PB 581	R THIGH, DONAR, D1, HS, THIC	950302	16.38	R THIGH/D1	0.170	0.588	0.417	0.417	0.154	KCL3-D1
PB 581	R THIGH, DONAR, HS, THICK A	950302	16.41	R THIGH/D2	0.080	0.750	0.667	0.333	0.246	KCL3-D2
PB 581	L THIGH, HS 1,FIRM	950302	16.42	L THIGH/HS	0.110	1.000	0.889	0.222	0.032	KCL3-1
PB 581	L THIGH, HS2	950302	16.43	L THIGH/HS	0.080	1.000	1.600	0.600	0.242	KCL3-2
PB 581	L THIGH, HS3	950302	16.44	L THIGH/HS	0.080	0.750	0.800	0.600	0.477	KCL3-3
PB 581	L THIGH,HS4	950302	16.45	L THIGH/HS	0.090	0.778	0.667	0.500	0.190	KCL3-3
PB 581	L THIGH, HS4	950302	16.45	L THIGH/HS	0.090	0.778	0.667	0.500	0.190	KCL3-4
PB 581	L KNEE, HS5, LATERAL ASPEC	950302	16.47	L KNEE/LAT	0.110	0.818	0.714	0.571	0.258	KCL3-5
PB 581	L KNEE, MEDIAL ASPECT, HS6	950302	16.49	L KNEE/MED	0.140	1.000	0.778	0.556	0.226	KCL3-6
PB 581	L ANKLE, LATERAL ASPECT, H	950302	16.50	L ANKLE/LA	0.100	0.700	0.571	0.429	0.233	KCL3-7
PB 581	L ANKLE, LAT MALLEOLUS, HS	950302	16.52	L ANK,HS8	0.070	0.714	0.750	0.750	0.299	KCL3-8
PB 581	L LEG, LATERAL ASPECT, HS9	950302	16.54	L LEG, LAT	0.090	0.778	1.000	0.500	0.287	KCL3-9
PB 581	HS1, L THIGH, ACTIVE	950420	16.37	HS1 L THI	0.120	0.750	0.556	0.333	0.202	KCL4-1
PB 581	R THIGH MEDIAL, NS VS HS1	950420	16.38	R THI NS	0.530	0.981	1.000	0.082	0.059	KCL4-1N
PB 581	HS2 L THIGH, LATERAL, ACTIV	950420	16.39	HS2, L THI	0.130	0.615	0.500	0.300	0.123	KCL4-2
PB 581	R THIGH, LAT.NS, VS KCL4-2	950420	16.40	R THI,NS	0.460	0.957	0.949	0.179	0.105	KCL4-2N
PB 581	L THIGH, HS3, ACTIVE	950420	16.41	HS3, L THI	0.120	0.750	0.875	0.500	0.380	KCL4-3
PB 581	NS, L THI, VS KCL4-3	950420	16.42	L THI,NS	0.370	0.946	1.062	0.156	0.114	KCL4-3N
PB 581	HS4, L THIGH, NEAR KNEE, AC	950420	16.44	HS4 L THI	0.090	0.667	0.667	0.500	0.251	KCL4-4
PB 581	R THIGH, ABOVE KNEE, NS VS	950420	16.45	NS L THI	0.500	0.960	1.022	0.111	0.091	KCL4-4N
PB 581	HS5, L KNEE LAT. ASPECT AC	950420	16.46	HS5 L KNEE	0.170	0.941	0.692	0.308	0.219	KCL4-5
PB 581	R KNEE, LAT NS VS KCL4-5	950420	16.48	R KNEE NS	0.310	0.903	0.923	0.192	0.118	KCL4-5N
PB 581	HS6 MED L KNEE, ACTIVE	950420	16.50	HS6 L KNEE	0.120	1.000	0.750	0.500	0.200	KCL4-6
PB 581	R KNEE MEDIAL, NS VS KCL4-6	950420	16.51	NS R KNEE	0.250	0.920	1.000	0.190	0.145	KCL4-6N
PB 581	L LEG, HS7, ACTIVE	950420	16.52	HS7 L LEG	0.130	0.846	0.700	0.300	0.147	KCL4-7
PB 581	R LEG, NS VS KCL4-7	950420	16.54	R LEG NS	0.200	0.850	0.813	0.250	0.165	KCL4-7N
PB 581	HS8, L ANKLE, LAT ASPECT	950420	16.55	HS8, L ANK	0.090	0.778	0.667	0.500	0.191	KCL4-8
PB 581	R ANKLE, NS VS KCL4-8	950420	16.56	R ANK,NS	0.490	1.000	1.194	0.361	0.236	KCL4-8N
PB 581	L CALF, HS9,FLAT	950420	16.57	HS9 L LEG	0.110	0.909	0.778	0.222	0.084	KCL4-9
PB 581	NS, R CALF VS KCL4-9	950420	16.58	R CALF	0.210	0.905	0.941	0.235	0.145	KCL4-9N
PB 581	HS10, L HIP, ACTIVE, SOFTER	950420	16.59	HS10 L HIP	0.190	0.842	0.769	0.462	0.289	KCL4-10
PB 581	NS, R HIP VS KCL4-10	950420	17.00	R HIP	0.400	0.975	1.057	0.143	0.089	KCL4-10N
PB 581	HS D1, R THIGH	950420	17.02	R THI,D1	0.150	0.867	0.900	0.500	0.305	KCL4-D1
PB581	HS D2, R THIGH	950420	17.03	R THI,D2	0.080	0.750	0.667	0.333	0.241	KCL4-D2
PB581	HS11, MATURE SG, L THIGH	950420	17.04	HS11 SG	0.270	0.963	1.000	0.174	0.129	KCL4-11
PB581	R THIGH, NS VS KCL4- D1, D2,	950420	17.06	R THI NS	0.430	0.977	1.026	0.132	0.084	KCL4-11N
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NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 581	HS1 L THIGH MEDIAL ASPECT	950610	11.49	HS1 L THI	0.160	0.875	0.909	0.455	0.136	KCL5-1
PB 581	HS2 L THIGH, MED	950610	11.51	HS2 L THI	0.130	0.769	0.600	0.300	0.278	KCL5-2
PB 581	HS3 L THIGH LATERAL	950610	11.52	HS3 L THI	0.060	1.000	0.800	0.200	0.116	KCL5-3
PB 581	HS4 L THIGH DISTAL	950610	11.53	HS4 L THI	0.130	0.692	0.750	0.625	0.280	KCL5-4
PB 581	HS5 L KNEE LATERAL	950610	11.54	HS5 L KNEE	0.100	0.900	0.875	0.250	0.084	KCL5-5
PB 581	HS6 L KNEE MEDIAL	950610	11.55	HS6 L KNEE	0.140	0.857	0.667	0.556	0.227	KCL5-6
PB 581	HS6 L KNEE MEDIAL	950610	11.55	HS6 L KNEE	0.140	0.857	0.667	0.556	0.227	KCL5-6
PB 581	HS7 L LEG, LATERAL, NEAR A	950610	11.57	HS7 L LEG	0.050	1.000	1.000	0.667	0.215	KCL5-7
PB 581	HS8 L ANKLE	950610	11.58	HS8 L ANK	0.110	0.818	0.500	0.375	0.200	KCL5-8
PB 581	HS9 L LEG, MID-SHAFT	950610	11.59	HS9 L LEG	0.170	0.765	0.538	0.308	0.266	KCL5-9
PB 581	HS10 L HIP	950610	12.00	HS10 L HIP	0.120	0.667	0.625	0.500	0.284	KCL5-10
PB 581	HS11 L THIGH MATURE MESHE	950610	12.09	HS11 L THI	0.240	0.917	0.750	0.200	0.128	KCL5-11
PB 581	D1 R THIGH,	950610	12.10	D1 R THI	0.090	0.778	0.571	0.286	0.278	KCL5-D1
PB 581	D2 R THIGH	950610	12.11	D2 R THI	0.110	0.909	0.625	0.375	0.310	KCL5-D2
PB 581	HS1 L THIGH, PROXIMAL	950713	16.56	HS1 L THI	0.180	0.722	0.643	0.286	0.156	KCL6-1
PB 581	HS2 L THIGH, LATERAL, PROXI	950713	16.57	HS1 L THI	0.160	1.000	0.583	0.333	0.176	KCL6-2
PB 581	HS3 L THIGH, MOST LATERAL	950713	16.58	HS1 L THI	0.140	0.786	0.600	0.400	0.169	KCL6-3
PB 581	HS4 L THIGH, DISTAL	950713	16.59	HS4 L THI	0.110	0.727	0.571	0.571	0.202	KCL6-4
PB 581	HS5 L KNEE, LATERAL	950713	17.00	HS5 L KNEE	0.110	0.818	0.750	0.375	0.222	KCL6-5
PB 581	HS6 L KNEE MEDIAL	950713	17.00	HS5 L KNEE	0.120	0.667	0.571	0.714	0.257	KCL6-6
PB 581	HS7 L LEG, ABOVE LAT. MALL	950713	17.01	HS7 L LEG	0.170	0.588	0.583	0.417	0.232	KCL6-7
PB 581	HS8 L ANKLE	950713	17.02	HS8 L ANK	0.240	0.583	0.444	0.333	0.236	KCL6-8
PB 581	HS9 L LEG, LATERAL BAND	950713	17.03	HS9 L LEG	0.230	0.957	0.842	0.211	0.129	KCL6-9
PB 581	HS10 L HIP	950713	17.04	HS10 L HIP	0.310	0.516	0.375	0.292	0.178	KCL6-10
PB 581	HS11 L THIGH, SSG, MATURE	950713	17.05	HS11 L SSG	0.360	0.917	0.781	0.125	0.081	KCL6-11
PB 581	D1 R THIGH, MIDDLE	950713	17.06	D1 R THI	0.160	0.813	0.545	0.455	0.202	KCL6-D1
PB 581	D2 R THIGH, MEDIAL	950713	17.07	D2 R THI	0.110	0.636	0.500	0.375	0.201	KCL6-D2
PB 581	HS1 L THIGH	950810	17.33	HS1 L THI	0.160	0.750	0.462	0.231	0.114	KCL7-1
PB 581	HS2 L THIGH	950810	17.34	HS1 L THI	0.180	0.722	0.643	0.286	0.216	KCL7-2
PB 581	HS3 L THIGH	950810	17.35	HS3 L THI	0.220	0.682	0.556	0.222	0.137	KCL7-3
PB 581	HS4 L THIGH, ABOVE KNEE	950810	17.35	HS4 L THI	0.060	1.000	1.000	0.500	0.190	KCL7-4
PB 581	HS5 L KNEE LATERAL	950810	17.36	HS5 K KNEE	0.140	0.643	0.500	0.400	0.199	KCL7-5
PB 581	HS6 L KNEE MEDIAL	950810	17.37	HS6 L KNEE	0.110	0.727	0.571	0.571	0.178	KCL7-6
PB 581	HS7 L LEG, ABOVE LAT MALL	950810	17.39	HS7 L LEG	0.090	0.667	0.667	0.500	0.277	KCL7-7
PB 581	HS8 L ANKLE, SSG	950810	17.40	HS8 L ANK	0.250	0.760	0.381	0.190	0.091	KCL7-8
PB 581	HS9 L LEG	950810	17.40	HS9 L LEG	0.210	0.714	0.611	0.167	0.125	KCL7-9
PB 581	HS10 L HIP	950810	17.41	HS10 L HIP	0.360	0.722	0.552	0.241	0.153	KCL7-10
PB 581	HS11 L THIGH, MSG,	950810	17.42	HS11 L THI	0.310	0.774	0.778	0.148	0.073	KCL7-11
PB 581	D1 R THIGH	950810	17.43	D1 R THIGH	0.160	0.750	0.583	0.333	0.205	KCL7-D1
PB 581	D2 R THIGH, MEDIAL ASPECT	950810	17.44	D2 R THIGH	0.100	0.800	0.625	0.250	0.161	KCL7-D2
PB 581	NS1 R THIGH	950810	17.45	NS1 R THI	0.460	0.957	0.952	0.095	0.054	KCL7-N1
PB 581	NS2 R KNEE	950810	17.46	NS2 R KNEE	0.460	0.957	0.976	0.122	0.059	KCL7-N2
PB 581	NS3 R LEG	950810	17.47	NS3 R LEG	0.210	0.905	0.833	0.167	0.128	KCL7-N3
PB 581	NS4 R ANKLE	950810	17.48	NS4 R ANK	0.310	0.677	0.654	0.192	0.097	KCL7-N4
PB 581	HS1 L THIGH	950907	16.56	HS1	0.180	1.000	1.000	0.286	0.192	KCL8-1
PB 581	HS2 L THIGH	950907	16.56	HS2	0.180	1.000	1.385	0.385	0.303	KCL8-2
PB 581	HS3 L THIGH, LATERAL	950907	16.57	HS3	0.180	1.000	0.643	0.286	0.224	KCL8-3
PB 581	HS4 L THIGH, DISTAL	950907	16.58	HS4	0.190	0.684	0.500	0.357	0.174	KCL8-4
PB 581	HS5 L KNEE, LATE4RAL	950907	16.59	HS5	0.240	0.833	0.789	0.263	0.148	KCL8-5
PB 581	HS6 L KNEE, MEDIAL	950907	16.59	HS6	0.100	0.700	0.571	0.429	0.241	KCL8-6
PB 581	HS7 L LEG, LATERAL	950907	17.00	HS7	0.110	1.000	0.625	0.375	0.138	KCL8-7
PB 581	HS8 L ANKLE	950907	17.01	HS8	0.150	0.733	0.700	0.500	0.233	KCL8-8
PB 581	HS9 L LEG BAND	950907	17.02	HS9	0.230	0.739	0.632	0.211	0.126	KCL8-9
PB 581	HS10 L HIP	950907	17.03	HS10	0.340	1.000	1.214	0.214	0.139	KCL8-10
PB 581	HS11 L THIGH, SSG	950907	17.04	HS11	0.250	0.840	0.714	0.190	0.141	KCL8-11
PB 581	D1 R THIGH	950907	17.04	D1	0.170	0.706	0.583	0.417	0.229	KCL8-D1
PB 581	D2 R THIGH, DONOR, MEDIAL	950907	17.05	D2	0.240	0.833	0.429	0.143	0.083	KCL8-D2
PB 581	HS1 L THIGH	951005	16.36	HS1 L THI	0.290	1.000	0.708	0.208	0.107	KCL9-1
PB 581	HS2	951005	16.38	HS1 L THI	0.270	1.000	1.286	0.286	0.205	KCL9-2
PB 581	HS3	951005	16.38	HS3	0.250	0.720	0.429	0.190	0.139	KCL9-3
PB 581	HS4	951005	16.39	HS4	0.190	0.579	0.538	0.462	0.244	KCL9-4
PB 581	HS5	951005	16.39	HS5	0.240	0.583	0.421	0.263	0.128	KCL9-5
PB 581	HS5	951005	16.39	HS5	0.240	0.583	0.421	0.263	0.128	KCL9-5
PB 581	HS6	951005	16.40	HS6	0.150	1.000	1.100	0.500	0.278	KCL9-6
PB 581	HS7	951005	16.41	HS7	0.120	0.750	0.750	0.500	0.340	KCL9-7
PB 581	HS8	951005	16.42	HS8	0.280	0.750	0.381	0.333	0.156	KCL9-8
PB 581	HS10	951005	16.43	HS10	0.370	0.757	0.548	0.194	0.106	KCL9-10
PB 581	HS9	951005	16.43	HS9	0.170	1.000	0.867	0.133	0.132	KCL9-9
PB 581	HS11	951005	16.44	HS11	0.210	0.810	0.529	0.235	0.141	KCL9-11
PB 581	D1	951005	16.44	D1	0.210	0.810	0.600	0.400	0.225	KCL9-D1
PB 581	D2	951005	16.45	D2	0.230	1.000	1.353	0.353	0.230	KCL9-D2

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 581	HS1 L THI,	951102	14.43	HS1 L THI	0.170	0.706	0.615	0.308	0.181	KCL10-1
PB 581	HS2 L THIGH	951102	14.44	L THI HS1	0.170	0.647	0.500	0.417	0.209	KCL10-2
PB 581	HS3 L THIGH, LATERAL	951102	14.44	L THI HS3	0.190	1.000	0.643	0.357	0.252	KCL10-3
PB 581	HS4 L THIGH, ABOVE KNEE	951102	14.45	L THI HS4	0.120	0.750	0.556	0.333	0.188	KCL10-4
PB 581	HS5 L KNEE, LATERAL	951102	14.46	HS5 L KNEE	0.150	1.000	0.800	0.500	0.268	KCL10-5
PB 581	HS6 L KNEE MEDIAL	951102	14.47	HS6 L KNEE	0.100	0.800	0.571	0.429	0.197	KCL10-6
PB 581	HS7 L LEG, LATERAL	951102	14.47	HS7 L LEG	0.210	1.000	0.471	0.235	0.134	KCL10-7
PB 581	HS8 L ANKLE, SSG	951102	14.48	HS8 L ANK	0.350	0.629	0.407	0.296	0.193	KCL10-8
PB 581	HS9 L LEG	951102	14.49	HS9 L LEG	0.240	1.000	1.263	0.263	0.179	KCL10-9
PB 581	HS10 L HIP	951102	14.50	HS10 L HIP	0.450	0.911	0.622	0.216	0.152	KCL10-10
PB 581	HS11 L THIGH, SSG, MATURE	951102	14.51	HS11 L THI	0.430	0.721	0.622	0.162	0.113	KCL10-11
PB 581	D1 R THIGH,	951102	14.51	D1 R THI	0.320	0.500	0.370	0.185	0.088	KCL10-D1
PB 581	D2 R THIGH	951102	14.52	D2 R THI	0.260	1.000	1.444	0.444	0.283	KCL10-D2
PB 581	HS1 L THI	951130	16.59	HS1M	0.240	0.833	0.722	0.333	0.177	KCL12-1
PB 581	HS2, L THI	951130	17.01	HS1M	0.210	0.810	0.588	0.235	0.166	KCL12-2
PB 581	HS3 L THI	951130	17.02	HS3	0.210	0.571	0.375	0.312	0.181	KCL12-3
PB 581	HS4 L THIGH, DISTAL	951130	17.02	HS4	0.260	0.769	0.478	0.130	0.116	KCL12-4
PB 581	HS5 L KNEE, MEDIAL	951130	17.03	HS5	0.180	0.500	0.385	0.385	0.190	KCL12-5
PB 581	HS6. L KNEE, LATERAL	951130	17.04	HS6	0.000	0.000	0.000	0.000	0.000	KCL12-6
PB 581	HS7 L LEG	951130	17.08	HS7 L LEG	0.140	1.000	1.556	0.556	0.257	KCL12-7
PB 581	HS8 L ANKLE	951130	17.09	HS8 L ANK	0.120	0.750	0.556	0.333	0.157	KCL12-8
PB 581	HS9 L CALF	951130	17.10	HS9	0.300	0.967	0.519	0.111	0.069	KCL12-9
PB 581	HS10 L HIP	951130	17.11	HS10 L HIP	0.220	0.955	0.706	0.294	0.160	KCL12-10
PB 581	D1 L THIGH	951130	17.11	D1	0.260	0.654	0.391	0.130	0.076	KCL12-D1
PB 581	D2 L THIGH	951130	17.12	D2	0.130	0.769	0.500	0.300	0.170	KCL12-D2
PB 581	HS1 L THIGH	951228	17.14	HS1 L THI	0.210	1.000	1.235	0.235	0.124	KCL13-1
PB 581	HS2 L THIGH	951228	17.15	HS2 L THI	0.160	1.000	1.143	0.143	0.093	KCL13-2
PB 581	HS3 L THIGH	951228	17.15	HS2 L THI	0.290	1.000	1.261	0.261	0.165	KCL13-3
PB 581	HS3 L THIGH	951228	17.15	HS3 L THI	0.290	1.000	1.261	0.261	0.165	KCL13-3
PB 581	HS4 L THIGH, DISTAL	951228	17.17	HS4	0.130	0.692	0.600	0.300	0.131	KCL13-4
PB 581	HS5 L KNEE, LATERAL	951228	17.17	HS5 L KNEE	0.290	0.724	0.478	0.261	0.175	KCL13-5
PB 581	HS6 L KNEE, MEDIAL	951228	17.18	HS6	0.130	0.923	0.625	0.625	0.290	KCL13-6
PB 581	HS7 L LEG,DISTAL	951228	17.19	HS7 L LEG	0.210	0.619	0.471	0.235	0.206	KCL13-7
PB 581	HS8 L ANKLE	951228	17.20	HS8 L ANK	0.190	0.632	0.438	0.187	0.105	KCL13-8
PB 581	HS10 L HIP	951228	17.21	HS10 L HIP	0.360	0.750	0.367	0.200	0.133	KCL13-10
PB 581	HS9 L LEG	951228	17.21	HS9 L LEG	0.190	1.000	1.357	0.357	0.211	KCL13-9
PB 581	HS11 L THIGH, MSG	951228	17.22	HS11 L THI	0.160	0.813	0.533	0.067	0.038	KCL13-11
PB 581	D1 R THIGH	951228	17.23	D1 R THI	0.160	1.000	1.231	0.231	0.174	KCL13-D1
PB 581	D2 R THIGH	951228	17.24	D2 R THI	0.270	1.000	1.227	0.227	0.118	KCL13-D2
PB 581	NS1 R THIGH	951228	17.25	NS1 R THI	0.630	0.794	0.759	0.086	0.063	KCL13-N1
PB 581	NS2 R HIP	951228	17.25	NS2 R HIP	0.400	0.975	0.972	0.111	0.066	KCL13-N2
PB 581	NS3 R KNEE MEDIAL	951228	17.26	NS3 R KNEE	0.550	1.000	0.958	0.146	0.083	KCL13-N3
PB 581	NS4 R KNEE LATERAL	951228	17.26	NS3 R KNEE	0.370	0.892	0.937	0.156	0.089	KCL13-N4
PB 581	NS5 R LEG	951228	17.27	NS5 R LEG	0.280	0.679	0.583	0.167	0.096	KCL13-N5
PB 581	NS6 R ANKLE	951228	17.28	NS6 R ANK	0.290	0.897	0.833	0.208	0.116	KCL13-N6
PB 581	HS1 L THIGH,MED	960125	16.44	HS1 L THI	0.350	0.629	0.323	0.129	0.053	KCL14-1
PB 581	HS2 L THIGH	960125	16.45	HS2	0.490	0.551	0.385	0.256	0.128	KCL14-2
PB 581	HS3 L THIGH	960125	16.46	HS3	0.240	0.875	0.556	0.333	0.199	KCL14-3
PB 581	HS4 L THIGH,DISTAL	960125	16.47	HS4	0.390	0.436	0.226	0.258	0.163	KCL14-4
PB 581	HS5 L KNEE LAT	960125	16.47	HS5	0.310	1.000	1.550	0.550	0.211	KCL14-5
PB 581	HS6 L KNEE MEDIAL	960125	16.48	HS6	0.410	0.439	0.371	0.171	0.054	KCL14-6
PB 581	HS7 L LEG	960125	16.48	HS7	0.220	0.364	0.353	0.294	0.216	KCL14-7
PB 581	HS8 L ANK	960125	16.49	HS8	0.270	0.556	0.400	0.350	0.194	KCL14-8
PB 581	HS9 L LEG	960125	16.49	HS9	0.330	1.000	0.808	0.269	0.158	KCL14-9
PB 581	HS10 L HIP	960125	16.50	HS10 L HIP	0.340	0.529	0.429	0.214	0.125	KCL14-10
PB 581	HS11 L THIGH, MESHED SG	960125	16.51	HS11 L THI	0.430	0.488	0.405	0.162	0.114	KCL14-11
PB 581	D1 R THIGH, DONOR	960125	16.51	D1 R THI	0.360	0.500	0.333	0.200	0.131	KCL14-D1
PB 581	D2 R THIGH	960125	16.52	D2 R THI	0.180	1.000	1.800	0.800	0.439	KCL14-D2
PB 581	NS1 R THIGH, MEDIAL, PROXI	960125	16.53	N1 R THI	0.630	0.794	0.804	0.125	0.068	KCL14-N1
PB 581	NS2 R KNEE MEDIAL	960125	16.53	N1 R KNEE	0.540	0.741	0.778	0.200	0.117	KCL14-N2
PB 581	NS3 R LEG	960125	16.54	N2 R LEG	0.300	1.000	1.000	0.304	0.183	KCL14-N3
PB 581	NS4 R ANKLE	960125	16.55	N4 R ANK	0.400	0.675	0.576	0.212	0.129	KCL14-N4

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 561	HS1	960222	14.07	HS1	0.000	0.000	0.000	0.000	0.000	KCL15-1
PB 561	HS2	960222	14.08	HS2	0.150	0.800	0.636	0.364	0.227	KCL15-2
PB 561	HS3	960222	14.08	HS3	0.130	0.769	0.800	0.300	0.118	KCL15-3
PB 561	HS4	960222	14.09	HS4	0.170	0.647	0.538	0.308	0.228	KCL15-4
PB 561	HS5	960222	14.09	HS5	0.170	0.706	0.545	0.545	0.318	KCL15-4
PB 561	HS5	960222	14.09	HS5	0.170	0.706	0.545	0.545	0.318	KCL15-5
PB 561	HS6	960222	14.10	HS6	0.280	0.393	0.333	0.556	0.240	KCL15-6
PB 561	HS7	960222	14.11	HS7	0.110	0.636	0.571	0.571	0.143	KCL15-7
PB 561	HS8	960222	14.11	HS8	0.330	0.788	0.615	0.269	0.123	KCL15-8
PB 561	HS10	960222	14.12	HS10	0.230	0.783	0.765	0.353	0.203	KCL15-10
PB 561	HS9	960222	14.12	HS9	0.160	0.688	0.692	0.231	0.199	KCL15-9
PB 561	D1	960222	14.13	D1	0.210	0.571	0.412	0.235	0.145	KCL15-D1
PB 561	D2	960222	14.13	D2	0.280	0.786	0.609	0.217	0.132	KCL15-D2
PB 581	HS1 L THIGH	960321	15.42	HS1	0.250	1.000	1.389	0.389	0.227	KCL16-1
PB 581	HS2 L THIGH	960321	15.42	HS2	0.250	1.000	1.389	0.389	0.227	KCL16-2
PB 581	HS1 L THIGH	960321	15.43	HS1	0.320	0.563	0.583	0.333	0.214	KCL16-1
PB 581	HS3 L THIGH	960321	15.44	HS3	0.410	0.707	0.606	0.242	0.151	KCL16-3
PB 581	HS4 L THIGH,DISTAL	960321	15.45	HS4	0.230	0.348	0.235	0.353	0.175	KCL16-4
PB 581	HS5 L KNEE MEDIAL	960321	15.45	HS5	0.210	0.571	0.467	0.400	0.225	KCL16-5
PB 581	HS6 L KNEE,LATERAL	960321	15.46	HS6	0.210	0.571	0.467	0.400	0.225	KCL16-6
PB 581	HS7 L LEG	960321	15.47	HS7	0.150	1.000	2.143	1.143	0.300	KCL16-7
PB 581	HS8 L FOOT	960321	15.47	HS8	0.330	0.364	0.208	0.375	0.172	KCL16-8
PB 581	HS10 L HIP	960321	15.49	HS10	0.310	0.645	0.500	0.292	0.175	KCL16-10
PB 581	HS9 L LEG BAND	960321	15.49	HS9	0.150	1.000	2.500	1.500	0.362	KCL16-9
PB 581	D1 R THIGH	960321	15.50	D1	0.360	0.333	0.194	0.161	0.096	KCL16-D1
PB 581	D2	960321	15.51	D1	0.330	0.485	0.407	0.222	0.157	KCL16-D2
PB 581	D2 inj	960321	15.51	Dinj	0.260	1.000	1.238	0.238	0.142	KCL16-Di
PB 581	ns r hip	960321	15.52	ns-hip	0.610	0.705	0.725	0.196	0.127	KCL16-n1
PB 581	ns r thigh,medial	960321	15.53	ns-thi	0.830	0.795	0.805	0.078	0.050	KCL16-n2
PB 581	ns r knee,lateral	960321	15.53	ns-knee	0.630	1.000	1.235	0.235	0.164	KCL16-n3
PB 581	ns r leg	960321	15.54	ns-leg	0.310	1.000	0.652	0.348	0.210	KCL16-n4
PB 581	ns r foot	960321	15.55	ns-leg	0.250	0.680	0.500	0.250	0.119	KCL16-n5
PB 518	HS1 L THIGH	960509	16.53	HS1 L THI	0.210	0.857	0.800	0.400	0.280	KCL17-1
PB 518	HS2 L THIGH	960509	16.54	HS2	0.160	0.813	0.571	0.143	0.077	KCL17-2
PB 518	HS3 L THIGH	960509	16.54	HS3	0.260	0.769	0.524	0.238	0.158	KCL17-3
PB 518	HS4 L THIGH DISTAL	960509	16.55	HS4	0.140	0.714	0.600	0.400	0.269	KCL17-4
PB 518	HS5 L KNEE, LATERAL	960509	16.56	HS5	0.280	0.750	0.542	0.167	0.114	KCL17-5
PB 518	HS6 L KNEE, MEDIAL	960509	16.57	HS6	0.150	0.800	0.909	0.364	0.259	KCL17-6
PB 518	HS6 L KNEE, MEDIAL	960509	16.57	HS6	0.150	0.800	0.909	0.364	0.259	KCL17-6
PB 518	HS7 L LEG	960509	16.58	HS7	0.130	1.000	1.286	0.857	0.696	KCL17-7
PB 518	HS7 L LEG	960509	16.58	HS7	0.130	1.000	1.286	0.857	0.696	KCL17-7
PB 518	HS8 L FOOT	960509	16.59	HS8	0.240	0.750	0.368	0.263	0.150	KCL17-8
PB 518	HS10 L HIP	960509	17.00	HS10	0.360	0.944	0.742	0.161	0.128	KCL17-10
PB 518	HS9 L CALF	960509	17.00	HS9	0.150	0.800	1.000	0.500	0.202	KCL17-9
PB 518	HS11 L THIGH, SSG	960509	17.01	HS11	0.210	0.905	0.765	0.235	0.111	KCL17-11
PB 518	D1 R THIGH	960509	17.02	D1	0.140	0.786	0.545	0.273	0.154	KCL17-D1
PB 518	D2 R THIGH	960509	17.03	D2	0.150	0.800	0.583	0.250	0.199	KCL17-D2
PB 518	D3 R THIGH, MEIDAL, INJ	960509	17.03	D3 inj	0.180	1.000	1.200	0.200	0.123	KCL17-D3
PB 581	HS1 L THIGH	960606	15.58	HS1	0.170	0.765	0.571	0.214	0.166	KCL18-1
PB 581	HS2 L THIGH	960606	15.59	HS2	0.220	1.000	1.056	0.222	0.116	KCL18-2
PB 581	HS3 L THIGH, POSTERIOR	960606	15.59	HS3	0.140	0.929	0.818	0.273	0.238	KCL18-3
PB 581	HS4 L THIGH, DISTAL	960606	16.00	HS4	0.130	0.923	0.800	0.300	0.248	KCL18-4
PB 581	HS5 L KNEE LATERAL	960606	16.01	HS5	0.180	0.889	0.733	0.200	0.100	KCL18-5
PB 581	HS6 L KNEE MEDIAL	960606	16.01	HS6	0.130	0.769	0.667	0.444	0.240	KCL18-6
PB 581	HS7 L LEG	960606	16.02	HS7	0.230	0.913	0.842	0.211	0.142	KCL18-7
PB 581	HS8 L ANKLE	960606	16.03	HS8	0.230	0.826	0.500	0.278	0.161	KCL18-8
PB 581	HS9 L LEG	960606	16.03	HS9	0.130	0.846	0.700	0.300	0.276	KCL18-9
PB 581	HS10 L HIP	960606	16.04	HS10	0.180	0.722	0.643	0.286	0.167	KCL18-10
PB 581	HS 11 L THIGH, MESHED SG	960606	16.04	HS11	0.270	0.963	0.913	0.174	0.093	KCL18-11
PB 581	D1 L THIGH	960606	16.05	D1	0.150	0.933	0.583	0.250	0.123	KCL18-D1
PB 581	D2 R THIGH, MEDIAL	960606	16.06	D2	0.300	0.867	0.760	0.200	0.145	KCL18-D2

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 624	HS1 L ARM	1/5/95	17.05	HS1 L ARM	0.120	0.917	0.889	0.333	0.143	LFY1
PB 624	HS2 L FOREARM, LATERAL AS	1/5/95	17.08	HS2 L FORA	0.140	0.643	0.778	0.556	0.331	LFY2
PB 624	HS3 L FOREARM, MEDIAL ASP	1/5/95	17.08	HS3 L FORA	0.180	0.778	0.714	0.286	0.113	LFY3
PB 624	NS R FOREARM MEDIAL ASPE	1/5/95	17.12	NS R FORAM	0.350	0.971	1.000	0.250	0.143	LFY6-NS
PB 624	NS R FOREARM, LATERAL ASP	1/5/95	17.13	NS R FORA	0.250	0.960	1.000	0.190	0.142	LFY5-NS
PB 624	NS R ARM, VS LFY1	1/5/95	17.15	NS R ARM	0.210	0.905	0.941	0.235	0.144	LFY4-NS
PB 624	HS3 L FOREARM,ACTIVE,MILD	3/16/95	15.48	HS3 L ARM	0.220	0.909	0.706	0.294	0.165	LFY3-3B
PB 624	HS3 L FOREARM,MEDIAL,2ND	3/30/95	15.20	HS3 L FORA	0.190	0.895	0.867	0.267	0.170	LFY4-3-2
PB 624	HS3 L FOREARM MEDIAL,3RD	3/30/95	15.21	HS3 L FORA	0.160	0.750	0.500	0.333	0.173	LFY4-3-3
PB 624	NS R FOREARM, VS LFY4-2	3/30/95	15.23	NS R FORAM	0.450	0.978	1.000	0.125	0.076	LFY4-2-N
PB 624	NS R ARM, VS LFY4-1	3/30/95	15.24	NS R ARM	0.290	1.000	1.040	0.160	0.101	LFY4-1-N
PB 624	NS R FOREARM,MEDIAL,VS LF	3/30/95	15.25	NS R FORA	0.370	0.946	1.000	0.156	0.100	LFY4-3-N
PB 628	L ARM, HS, 3/12 POST-INJURY,	950216	15.43	L ARM-HS1	0.210	0.857	0.929	0.500	0.376	LFY2-1
PB 628	L FOREARM, ACTIVE HS, 3/12	950216	15.45	L FOREARM	0.230	0.739	0.933	0.533	0.339	LFY2-2
PB 628	L FOREARM MEDIAL ASPECT,	950216	15.46	L FOREARM	0.130	0.769	0.556	0.444	0.257	LFY2-3
PB 624	L ELBOW, HS1, ACTIVE, 4/12 A	950316	15.39	L ELB HS1	0.190	0.789	0.615	0.462	0.217	LFY3-1
PB 624	L ELBOW, HS1, 2ND MEASURE	950316	15.41	L ELB HS1	0.180	0.778	0.571	0.286	0.169	LFY3-1B
PB 624	L ELBOW, HS2, ACTIVE SCAR	950316	15.43	L ELB HS2	0.150	0.800	0.800	0.500	0.281	LFY3-2
PB 624	L ELBOW HS2, 2ND MEASURE	950316	15.45	L ELB HS2	0.170	0.824	0.909	0.545	0.371	LFY3-2B
PB 624	L FOREARM HS3, HS, MILDLY	950316	15.46	L ARM HS3	0.180	0.778	0.571	0.286	0.164	LFY3-3
PB 624	L ARM, HS1, ACTIVE, FIRM,	950330	15.09	L ELB, HS1	0.210	0.857	0.800	0.400	0.266	LFY4-1-1
PB 624	L ARM, HS1, 2ND MEASUREME	950330	15.11	L ELB, HS1	0.000	0.000	0.000	0.000	0.000	LFY4-1-2
PB 624	L ARM, HS1, 3RD MEASUREME	950330	15.13	L ELB, HS1	0.160	0.813	0.833	0.333	0.194	LFY4-1-3
PB 624	L FOREARM, HS2, ACTIVE, FIR	950330	15.14	L ELB, HS1	0.180	0.722	0.583	0.500	0.303	LFY4-1-3
PB 624	L FOREARM ACTIVE, FIRM HS2	950330	15.14	L ELB, HS1	0.180	0.722	0.583	0.500	0.303	LFY4-2-1
PB 624	L FOREARM, HS2, 2ND MEASU	950330	15.16	L ELB, HS1	0.190	0.842	0.692	0.462	0.234	LFY4-2-2
PB 624	L FOREARM, HS2, 3RD MEASU	950330	15.17	L ELB, HS1	0.170	0.706	0.636	0.545	0.333	LFY4-2-3
PB 624	L FOREARM, MEDIAL HS3	950330	15.18	L ELB, HS1	0.190	0.842	0.733	0.267	0.168	LFY4-3-1
PB 624	R FOREARM, NS, VS HS2	950330	15.22	R FOREARM	0.450	0.978	1.000	0.125	0.076	LFY4-2-N
PB 624	L ARM, HS1, VERY ACTIVE, RE	950420	15.51	HS1,L ARM	0.100	0.800	0.714	0.429	0.173	LFY5-1
PB 624	HS2, L FOREARM, ACTIVE, RE	950420	15.53	HS2 L FORA	0.170	0.706	0.667	0.417	0.237	LFY5-2
PB 624	HS3, L MEDIAL FOREARM, ACT	950420	15.54	HS3 L FORA	0.150	0.800	0.667	0.250	0.113	LFY5-3
PB 624	R ARM, NS VS LFY5-1	950420	15.55	R ARM,NS	0.540	0.963	1.021	0.125	0.082	LFY5-1N
PB 624	R FOREARM, NS VS LFY5-2	950420	15.56	R FORA,NS	0.340	0.941	1.000	0.214	0.138	LFY5-2N
PB 624	R MEDIAL FOREARM NS VS LF	950420	15.57	R ME FOR	0.440	0.977	1.081	0.189	0.104	LFY5-3N
PB 624	HS1 L ARM, ACTIVE	950504	16.19	HS1 L ARM	0.100	1.000	0.875	0.250	0.124	LFY4-1
PB 624	HS2 L ELBOW, ACTIVE	950504	16.20	HS2 L ELB	0.190	0.789	0.786	0.357	0.237	LFY4-2
PB 624	HS3, L FOREARM, LATERAL	950504	16.21	HS3 L FOM	0.180	0.778	0.692	0.385	0.187	LFY4-3
PB 624	HS4 L FOREARM MEDIAL	950504	16.22	HS4 L FO M	0.140	0.857	0.900	0.400	0.248	LFY4-4
PB 624	NS VS LFY4-1	950504	16.23	R ARM, NS	0.330	0.909	1.125	0.375	0.213	LFY4-1N
PB 624	NS, VS LFY4-2	950504	16.24	R ELB,NS	0.280	0.821	0.840	0.120	0.092	LFY4-2N
PB 624	NS VS LFY4-3	950504	16.25	R FORM,NS	0.290	0.931	1.042	0.208	0.148	LFY4-3N
PB 624	NS, VS LFY4-4	950504	16.27	R FORM,M	0.360	0.944	1.067	0.200	0.120	LFY4-4N
PB 624	HS1 L ARM	950601	15.49	HS1 L ARM	0.180	0.556	0.462	0.385	0.207	LFY6-1
PB 624	HS2 L ELBOW	950601	15.49	HS2 L ELB	0.350	0.829	0.741	0.296	0.191	LFY6-2
PB 624	HS3 L FOREARM	950601	15.50	HS3 L FORA	0.190	0.842	0.733	0.267	0.163	LFY6-3
PB 624	NS1 R ARM	950601	15.51	NS1 R ARM	0.310	0.935	1.038	0.192	0.146	LFY6-1N
PB 624	NS2 R ELBOW	950601	15.52	NS2 R ELB	0.520	0.942	0.957	0.106	0.073	LFY6-2N
PB 624	NS3 R FOREARM	950601	15.53	NS3 R FORA	0.490	0.959	1.047	0.140	0.086	LFY6-3N

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
BO 650	HS1 L ARM	12/31/94	11.45	HS1 L ARM	0.160	0.813	0.727	0.455	0.227	LSF1
BO 650	HS2 L FOREARM	12/31/94	11.47	HS2 L FORM	0.140	0.786	0.700	0.400	0.221	LSF2
BO 660	HS1 L ARM,13/12 POST-INJURY	4/29/95	11.50	HS1 L ARM	0.190	0.842	0.600	0.267	0.183	LSF4-1B
BO 650	L ARM, ACTIVE HS, IMPROVED	950216	16.34	L ARM, HS	0.140	0.929	0.889	0.556	0.314	LSF2-1
BO 650	L FOREARM, ACTIVE HS, IMPR	950216	16.35	L FOREARM	0.160	0.813	0.727	0.455	0.271	LSF2-2
BO 650	L ARM, HS1, ACTIVE	950316	16.38	L ARM HS1	0.240	0.792	0.556	0.333	0.158	LSF3-1
BO 650	L FOREARM, HS2, ACTIVE, FIR	950316	16.40	L FORE HS2	0.130	0.769	0.600	0.300	0.167	LSF3-2
BO 650	HS1 L ARM, 13/12 POST INJUR	950429	11.48	HS1 L ARM	0.230	0.826	0.556	0.278	0.140	LSF4-1
BO 650	HS2 L FOREARM,	950429	11.50	HS2 L FORA	0.150	0.733	0.636	0.364	0.140	LSF4-2
BO 650	HS2 L FOREARM,	950429	11.50	HS2 L FORA	0.150	0.733	0.636	0.364	0.140	LSF4-2
BO 650	L FOREARM, HS2, 2ND MX	950429	11.52	HS2 L FORA	0.110	0.818	0.714	0.571	0.262	LSF4-2B
BO 650	R ARM, NS	950429	11.55	NS R ARM	0.370	0.973	1.097	0.194	0.117	LSF4-1N
BO 650	R ARM, NS	950429	11.56	NS R ARM	0.300	0.967	1.042	0.250	0.182	LSF4-1NB
BO 650	NS, R FOREARM	950429	11.57	NS R FORAM	0.350	0.943	1.033	0.167	0.140	LSF4-2N
BO 650	R FOREARM, NS VS LSF4-2	950429	11.57	NS R FORAM	0.340	0.971	1.071	0.214	0.119	LSF4-2NB
BO 650	HS1 L SHOULDER	950506	10.53	HS1 L SHO	0.160	0.750	0.667	0.333	0.186	LSF1-S1
BO 650	HS1 L SHOULDER	950506	12.58	HS1 L SHO	0.110	1.000	0.857	0.571	0.333	LSF1-S2
BO 641	HS1 L SHOULDER	5/6/95	12.59		0.110	1.000	0.857	0.571	0.333	LSF1-S2
BO 650	HS1 L SHOULDER	950506	13.00	HS1 L SHO	0.120	0.750	0.857	0.714	0.379	LSF1-S3
BO 641	HS1 L SHOULDER	5/6/95	13.01		0.120	0.750	0.857	0.714	0.379	LSF1-S3
BO 650	HS2 L ARM MEDIAL	950506	10.56	HS2 MED	0.200	0.800	0.600	0.333	0.188	LSF2-S1
BO 650	HS2 L ARM MEDIAL	950506	13.02	HS2 L MED	0.190	0.842	0.643	0.357	0.253	LSF2-S2
BO 641	HS2 L ARM MEDIAL	5/6/95	13.03		0.190	0.842	0.643	0.357	0.253	LSF2-S2
BO 650	HS2 L ARM MEDIAL	950506	13.04	HS2 L MED	0.200	0.900	0.688	0.250	0.204	LSF2-S3
BO 641	HS2 L ARM MEDIAL	5/6/95	13.05		0.200	0.900	0.688	0.250	0.204	LSF2-S3
BO 650	HS3 L ARM LATERAL ASPECT	950506	10.59	HS2 LAT	0.140	0.714	0.500	0.400	0.206	LSF3-S1
BO 650	HS2 L ARM LATERAL	5/6/95	13.09		0.160	0.875	0.667	0.333	0.212	LSF3-S2
BO 650	HS3 L ARM LATERAL	950506	13.09	HS3 L LAT	0.160	0.875	0.667	0.333	0.212	LSF3-S2
BO 650	HS3 L ARM LATERAL	5/6/95	13.11		0.220	0.818	0.667	0.222	0.118	LSF3-S3
BO 650	HS3 L ARM LATERAL	950506	13.11	HS3 L LAT	0.220	0.818	0.667	0.222	0.118	LSF3-S3
BO 650	HS4 L FOREARM	950506	11.01	HS4 L FORA	0.130	0.769	0.667	0.444	0.229	LSF4-S1
BO 650	HS4 L FOREARM	950506	13.13	HS4 L FORM	0.100	0.800	0.714	0.429	0.174	LSF4-S2
BO 650	HS4 L FOREARM	5/6/95	13.14	HS4 L FORE	0.100	0.800	0.714	0.429	0.174	LSF4-S2
BO 650	HS4 L FOREARM	5/6/95	13.15	HS4 L FORE	0.110	0.818	0.750	0.375	0.201	LSF4-S3
BO 650	HS4 L FOREARM	950506	13.15	HS4 L FORM	0.110	0.818	0.750	0.375	0.201	LSF4-S3
BO 650	HS1 L ARM, MEDIAL	950701	11.11	HS1 L ARM	0.240	0.875	0.765	0.412	0.220	LSF6-1
BO 650	HS2 L ARM, LATERAL	950701	11.12	HS2 L ARM	0.180	0.889	0.786	0.286	0.189	LSF6-2
BO 650	HS3 L FOREARM	950701	11.13	HS3 L FORA	0.220	0.864	0.647	0.294	0.172	LSF6-3
BO 650	NS1 R ARM	950701	11.14	NS1 R ARM	0.440	0.955	1.026	0.158	0.118	LSF6-1NS
BO 650	NS2 R FOREARM,	950701	11.14	NS2 R FORA	0.470	0.957	1.024	0.146	0.101	LSF6-2NS
BO 650	HS1 L ARM,	950819	11.24	HS1 L ARM	0.310	1.000	0.769	0.192	0.129	LSF7-1
BO 650	HS1 L ARM, 2ND MX	950819	11.26	HS1 L ARM	0.230	0.783	0.526	0.211	0.109	LSF7-1B
BO 650	HS2 L FOREARM	950819	11.28	HS2 L FORA	0.180	0.778	0.571	0.286	0.179	LSF7-2
BO 650	HS2 L FOREARM, 2ND MX	950819	11.28	HS2 L FORA	0.210	0.810	0.625	0.312	0.229	LSF7-2B
BO 650	NS1 R ARM	950819	11.29	NS1 R ARM	0.390	0.949	0.943	0.114	0.087	LSF7-1NS
BO 650	NS2 R FOREARM	950819	11.30	NS1 R ARM	0.380	0.947	0.941	0.118	0.074	LSF7-2NS
BO 650	HS1 L ARM, PALER, SOFTER	951028	11.08	HS1 L ARM	0.520	0.788	0.467	0.156	0.100	LSF8-1
BO 650	HS2 L FOREARM, PALER, STIL	951028	11.09	HS2 L FORE	0.250	0.760	0.476	0.190	0.111	LSF8-2
BO 650	NS1 R ARM	951028	11.10	NS1 R ARM	0.510	1.000	1.133	0.133	0.077	LSF8-1NS
BO 650	NS2 R FOERARM	951028	11.11	NS2 R FORA	0.430	0.977	0.789	0.132	0.104	LSF8-2NS
BO 650	HS1 L ARM	951125	11.24	HS1 L ARM	0.220	0.727	0.412	0.294	0.151	LSF9-1
BO 650	HS1 L ARM, 2ND MX	951125	11.24	HS1 L ARM	0.260	0.731	0.429	0.238	0.158	LSF9-1B
BO 650	HS2 L FOREARM	951125	11.25	HS2 L FORM	0.120	0.750	0.625	0.500	0.207	LSF9-2
BO 650	HS2 L FOREARM, 2ND MX	951125	11.26	HS2 L FORM	0.190	0.789	0.500	0.187	0.093	LSF9-2B
BO 650	NS1 R ARM	951125	11.27	NS1 R ARM	0.580	0.828	0.827	0.115	0.077	LSF9-1N
BO 650	NS1 R ARM, 2ND MX	951125	11.28	NS1 R ARM	0.600	0.933	0.839	0.071	0.067	LSF9-1NB
BO 650	NS2 R FOREARM	951125	11.28	NS2 R FORM	0.590	0.797	0.759	0.093	0.069	LSF9-2N
BO 650	NS2 R FOREARM, 2ND MX	951125	11.29	NS2 R FORM	0.460	0.957	0.929	0.095	0.083	LSF9-2NB
BO 650	HS1 L ARM	951223	11.39	HS1 L ARM	0.250	0.840	0.579	0.316	0.164	LSF10-1
BO 650	HS1 L ARM, 2ND MX	951223	11.39	HS1 L ARM	0.320	0.875	0.538	0.231	0.123	LSF10-1B
BO 650	HS2 L FOREARM	951223	11.41	HS2 L FORM	0.200	0.750	0.643	0.429	0.241	LSF10-1
BO 650	HS2 L FOREARM	951223	11.41	HS2 L FORM	0.200	0.750	0.643	0.429	0.241	LSF10-2
BO 650	HS2 L FOREARM, 2ND MX	951223	11.42	HS2 L FORM	0.200	0.700	0.600	0.333	0.237	LSF10-2
BO 650	HS2 L FOREARM, 2ND MX	951223	11.42	HS2 L FORM	0.200	0.700	0.600	0.333	0.237	LSF10-2B
BO 650	NS1 R ARM	951223	11.43	NS1 R ARM	0.450	0.978	1.050	0.125	0.093	LSF10-N1
BO 650	NS2 R FOREARM	951223	11.44	NS2 R FORM	0.440	0.955	0.974	0.128	0.099	LSF10-N2
BS 570	HS1 L ARM	960120	11.33	HS1 L ARM	0.270	0.741	0.455	0.227	0.105	LSF11-1
BO 650	HS2 L FOREARM	960120	11.34	HS2 L FORM	0.200	0.800	0.667	0.333	0.210	LSF11-2
BO 650	NS1 R ARM	960120	11.36	NS1 R ARM	0.550	0.964	0.941	0.078	0.058	LSF11-N1
BO 650	NS2 R FOREARM	960120	11.37	NS2 R FORM	0.430	0.884	0.846	0.103	0.061	LSF10-N2
BO 650	NS2 R FOREARM	960120	11.37	NS2 R FORM	0.430	0.884	0.846	0.103	0.061	LSF11-N2

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NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
BO 682	HS1 R FOOT, ACTIVE, 2/12 PO	950429	11.23	HS1 R FOOT	0.130	0.846	0.900	0.300	0.152	LSL1-1
BO 682	NS, L FOOT, VS LSL1-1	950429	11.26	NS L FOOT	0.250	0.920	0.900	0.250	0.190	LSL1-1N
BO 682	R FOOT, HS1 ACTIVE, 2ND MX	950429	11.27	HS1 R FOOT	0.060	0.833	0.600	0.200	0.145	LSL1-1B
BO 682	HS1 R FOOT, ACTIVE 3RD MX	950429	11.29	HS1 R FOOT	0.100	0.800	0.857	0.429	0.290	LSL1-1C
BO 682	HS2, R KNEE, MED, ACTIVE	950429	11.30	HS2 R KNEE	0.160	0.813	0.917	0.333	0.207	LSL1-2
BO 682	HS3 R THIGH MEDIAL, ACTIVE	950429	11.31	HS3 R THI	0.210	0.857	0.875	0.312	0.191	LSL1-3
BO 682	R THIGH, MEDIAL, PROXIMAL,	950429	11.32	HS4 R THI	0.410	0.951	1.029	0.206	0.138	LSL1-4
BO 682	HS5, L KNEE MED, ACTIVE	950429	11.33	HS5 L KNEE	0.170	0.824	0.769	0.308	0.165	LSL1-5
BO 682	NS, L THIGH, VS LSK2-5	950429	11.34	NS L THI	0.320	0.969	1.115	0.231	0.145	LSL1-5
BO 682	NS L THIGH, VS LSL1-2-5	950429	11.34	NS L THI	0.320	0.969	1.115	0.231	0.145	LSL1-5N
BO 682	L KNEE, HS5, ACTIVE	950429	11.36	HS5 L KNEE	0.130	1.000	0.727	0.182	0.119	LSL1-5
BO 682	L KNEE, HS5, 2ND MX	950429	11.37	HS5 L KNEE	0.130	0.769	0.667	0.444	0.269	LSL1-5B
BO 682	HS3 R THIGH MED, 2ND MX	950429	11.39	HS3 R THI	0.230	0.913	0.895	0.211	0.126	LSL1-3B
BO 682	R THIGH, HS4, PROXIMAL, 2ND	950429	11.40	HS4 R THI	0.380	0.947	1.065	0.226	0.157	LSL1-4B
BO 682	HS1 R ANKLE	950601	17.16	HS1 R ANK	0.140	0.857	0.818	0.273	0.112	LSL2-1
BO 682	HS2 L KNEE	950601	17.18	HS2 R KNEE	0.180	0.944	0.867	0.200	0.103	LSL2-2
BO 682	HS3 R THIGH, MIDDLE	950601	17.19	HS2 R KNEE	0.200	0.950	1.067	0.333	0.219	LSL2-3
BO 682	HS3 MED-R THIGH	950601	17.19	HS3 R THI	0.200	0.950	1.067	0.333	0.219	LSL2-3
BO 682	HS4 PROXIMAL R THIGH	950601	17.20	HS4 R THI	0.370	0.973	1.032	0.194	0.125	LSL2-4
BO 682	HS5 L THIGH, MEDIAL	950601	17.21	HS5 L THI	0.200	1.000	1.267	0.333	0.194	LSL2-5
BO 682	HS6 L KNEE	950601	17.22	HS6 L KNEE	0.220	0.818	0.882	0.294	0.161	LSL2-6
BO 682	NS L THIGH	950601	17.23	NS L THI	0.240	0.958	1.105	0.263	0.146	LSL2-1NS
BS 555	HS1 R LEG	950629	16.47	HS1 R LEG	0.140	0.857	0.727	0.273	0.160	LSL3-1
BO 682	HS2 R KNEE MEDIAL	950629	16.48	HS2 R KNEE	0.300	0.867	0.875	0.250	0.139	LSL3-2
BO 682	HS3 R THIGH	950629	16.49	HS3 R THI	0.250	0.920	1.000	0.190	0.119	LSL3-3
BO 682	HS4 L THIGH	950629	16.50	HS4 L THI	0.230	0.870	0.889	0.278	0.141	LSL3-4
BO 682	NS1 L LEG	950629	16.51	NS1 L LEG	0.330	0.939	1.000	0.269	0.175	LSL3-1NS
BO 682	NS2 L THIGH	950629	16.52	NS2 L THI	0.380	0.974	1.030	0.152	0.123	LSL3-2NS
BO 682	HS1 R THIGH MEDIAL	950727	17.17	HS1 R THI	0.250	0.800	0.650	0.250	0.159	LSL4-1
BO 682	HS2 L KNEE MEDIAL	950727	17.18	HS2 L KNEE	0.290	0.862	0.833	0.208	0.107	LSL4-2
BO 682	HS3 R FOOT	950727	17.20	HS3 R FOOT	0.150	0.867	0.833	0.250	0.191	LSL4-3
BO 682	HS1 R LEG	950907	17.30	HS1 R LEG	0.170	0.824	0.769	0.308	0.177	LSL5-1
BO 682	HS2 R KNEE, MEDIAL	950907	17.31	HS2	0.320	0.813	0.667	0.185	0.109	LSL5-2
BO 682	HS3 R THIGH, DISTAL	950907	17.32	HS3	0.310	0.806	0.731	0.192	0.114	LSL5-3
BO 682	HS4 R THIGH	950907	17.32	HS4	0.340	0.882	0.933	0.133	0.074	LSL5-4
BO 682	HS5 L THIGH	950907	17.33	HS5	0.310	0.645	0.630	0.148	0.093	LSL5-5
BO 682	HS6 L KNEE, MEDIAL	950907	17.34	HS6	0.250	0.760	0.632	0.316	0.174	LSL5-6
BO 682	HS1 R LEG, MATURE	951005	17.18	HS1 R LEG	0.290	0.759	0.667	0.208	0.157	LSL6-1
BO 682	HS2 R KNEE, MEDIAL, ANGRY	951005	17.19	HS2 R KNEE	0.340	0.706	0.552	0.172	0.144	LSL6-2
BO 682	HS3 R THIGH, DISTAL	951005	17.20	HS2 R KNEE	0.350	0.657	0.548	0.129	0.104	LSL6-3
BO 682	HS4 R THIGH	951005	17.20	HS2 R KNEE	0.530	1.000	1.082	0.082	0.073	LSL6-4
BO 682	HS5 L THIGH	951005	17.21	HS2 R KNEE	0.350	1.000	0.800	0.167	0.161	LSL6-5
BO 682	HS6 L KNEE MEDIAL	951005	17.21	HS6 L KNEE	0.240	0.750	0.611	0.333	0.177	LSL6-6
BO 682	HS1 R LEG	951102	15.24	HS1 R LEG	0.200	0.700	0.625	0.250	0.144	LSL7-1
BO 682	NS1 L LEG	951102	15.25	NS1 L LEG	0.270	0.778	0.667	0.125	0.093	LSL7-1N
BO 682	HS2 R KNEE	951102	15.26	HS2 R KNEE	0.250	0.840	0.750	0.250	0.171	LSL7-2
BO 682	HS3 R THIGH DISTAL	951102	15.26	HS3 R THI	0.380	0.684	0.606	0.152	0.124	LSL7-3
BO 682	HS4 R THIGH	951102	15.27	HS4 R THI	0.380	1.000	0.771	0.086	0.052	LSL7-4
BO 682	HS5 L THIGH	951102	15.28	HS5 L THI	0.350	0.914	0.867	0.167	0.109	LSL7-5
BO 682	HS5 L THIGH	951102	15.28	HS6 L KNEE	0.350	0.914	0.867	0.167	0.109	LSL7-5
BO 682	HS6 L KNEE	951102	15.28	HS6 L KNEE	0.350	0.914	0.867	0.167	0.109	LSL7-6
BO 682	HS1 R KNEE, MEDIAL	951130	16.40	HS1 R KNEE	0.320	0.844	0.704	0.185	0.131	LSL8-1
BO 682	HS2 L THIGH	951130	16.41	HS2 L THI	0.470	0.894	0.881	0.119	0.084	LSL8-2
BO 682	HS3 L KNEE	951130	16.41	HS2 L THI	0.400	0.875	0.778	0.111	0.068	LSL8-3
BO 682	HS1 R LEG	951228	17.59	HS1 R LEG	0.190	0.789	0.600	0.267	0.147	LSL9-1
BO 682	HS2 R KNEE, MEDIAL	951228	18.00	HS2 R KNEE	0.470	0.872	0.738	0.119	0.070	LSL9-2
BO 682	HS3 R THIGH, DISTAL	951228	18.01	HS3 R THI	0.280	1.000	1.217	0.217	0.138	LSL9-3
BO 682	HS4 R THIGH, PROXIMAL	951228	18.01	HS4 R THI	0.560	1.000	0.843	0.098	0.069	LSL9-4
BO 682	HS5 L THIGH	951228	18.02	HS5 L THI	0.380	1.000	0.886	0.086	0.057	LSL9-5
BO 682	HS6 L KNEE	951228	18.03	HS6 L KNEE	0.320	0.844	0.714	0.143	0.093	LSL9-6
BO 682	NS1 L THIGH,MEDIAL	951228	18.03	NS1 L THI	0.490	0.776	0.727	0.114	0.069	LSL9-N1
BO 682	HS1 R LEG	960125	17.35	HS1 R LEG	0.280	0.607	0.619	0.333	0.205	LSL10-1
BO 682	NS1 L LEG	960125	17.36	NS1 L LEG	0.370	0.595	0.452	0.194	0.124	LSL10-N1
BO 682	HS2 R KNEE	960125	17.37	HS2 R KNEE	0.500	0.560	0.477	0.136	0.077	LSL10-2
BO 682	HS3 R THIGH,DISTAL	960125	17.37	HS3 R THI	0.410	1.000	0.857	0.171	0.117	LSL10-3
BO 682	HS4 R THIGH	960125	17.38	HS4 R THI	0.420	0.929	0.806	0.167	0.099	LSL10-4
BO 682	HS5 L THIGH	960125	17.39	HS5 L THI	0.340	1.000	0.759	0.172	0.097	LSL10-5
BO 682	HS6 L KNEE MEDIAL	960125	17.40	HS6 L KNEE	0.440	0.841	0.811	0.189	0.113	LSL10-6
BO 682	NS2 L THIGH,MEDIAL	960125	17.41	NS2 L THI	0.640	0.797	0.724	0.103	0.060	LSL10-N2

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NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 588	HS1 R THIGH, AFTER INJECTIO	1/6/96	12.09	HS2 R THI	0.220	1.000	1.158	0.158	0.091	LYY10-1A
PB 588	HS1 R THIGH, PROXIMAL	5/18/96	11.27	HS2 R inj	0.130	0.692	0.556	0.444	0.228	LYY14-1
PB 588	R THIGH 1, HS ALONG RIM OF	950311	11.01	R THIGH/HS	0.250	0.840	0.524	0.190	0.157	LYY3-1
PB 588	MATURE SSG, ON R THIGH, 13/	950311	11.03	R THIGH/SG	0.140	0.857	0.700	0.400	0.187	LYY3-2
PB 588	MATURE DONAR SITE, NOT HY	950311	11.04	L THIGH/SG	0.330	0.939	0.931	0.138	0.096	LYY3-3
PB 588	NORMAL SKIN ON R THIGH VS	950311	11.05	R THIGH/NS	0.420	0.976	1.000	0.135	0.116	LYY3-5
PB 588	R THIGH HS4, FIRM, RAISED, P	950311	11.07	R THIGH/HS	0.110	0.727	0.500	0.375	0.141	LYY3-4
PB 588	NS1 R THIGH	950810	15.50	NS1 R THI	0.480	0.958	0.955	0.091	0.081	LYY4-1
PB 588	NS1 R THIGH,	950810	15.50	NS1 R THI	0.480	0.958	0.955	0.091	0.081	LYY4-1NS
PB 588	HS1 R THIGH, LATERAL	950810	15.52	HS1 R THI	0.330	0.909	0.875	0.375	0.192	LYY4-1
PB 588	HS1 R THIGH, LATRAL, 2ND MX	950810	15.53	HS1 R THI	0.360	0.750	0.710	0.161	0.094	LYY4-1B
PB 588	HS2 R THIGH, MATURE SSG	950810	15.54	HS1 R THI	0.310	0.871	0.778	0.148	0.083	LYY4-2
PB 588	HS2 R THIGH, SSG MATURE	950810	15.55	HS1 R THI	0.260	0.885	0.783	0.130	0.068	LYY4-2B
PB 588	HS3 L THIGH, DONOR	950810	15.55	HS3 L THI	0.400	0.925	0.946	0.081	0.069	LYY4-3
PB 588	HS3 L THIGH, DONOR	950810	15.56	HS3 L THI	0.460	0.935	0.884	0.070	0.039	LYY4-3B
PB 588	HS4 L THIGH, ABOVE KNEE	950810	15.57	HS4 L THI	0.180	0.722	0.500	0.286	0.127	LYY4-4
PB 588	HS4 L THIGH, ABOVE KNEE, 2	950810	15.57	HS4 L THI	0.130	0.692	0.556	0.444	0.171	LYY4-4B
PB 588	HS1 R THIGH, SSG, PROXIMAL	950916	10.51	HS1 R THI	0.290	1.000	0.833	0.208	0.155	LYY5-1
PB 588	HS2 R THIGH, SSG, DISTAL	950916	10.52	HS2 R THI	0.190	0.895	0.667	0.267	0.149	LYY5-2
PB 588	HS3 L THIGH, DONOR, MATUR	950916	10.53	HS3 L THI	0.370	0.946	0.970	0.121	0.066	LYY5-3
PB 588	HS4 L THIGH, DISTAL	950916	10.54	HS4 L THI	0.300	0.767	0.522	0.304	0.183	LYY5-4
PB 588	NS R THIGH, MEDIAL	950916	10.54	NS R THI	0.600	0.950	0.857	0.071	0.060	LYY5-NS
PB 588	HS1 R THIGH, SSG, PROXIMAL	951014	11.31	HS1 R THI	0.270	0.778	0.636	0.227	0.170	LYY7-1
PB 588	HS2 R THIGH, RIM OF SSG	951014	11.32	HS2 R THI	0.230	0.870	0.611	0.278	0.129	LYY7-2
PB 588	HS3 L THIGH, DONOR, MATUR	951014	11.33	HS3 L THI	0.500	0.940	0.851	0.064	0.053	LYY7-3
PB 588	HS4 L THIGH, DISTAL	951014	11.33	HS4 L THI	0.240	0.792	0.412	0.412	0.165	LYY7-4
PB 588	S5 R THIGH, LATERAL SCAR A	951014	11.34	HS5 R THI	0.450	1.000	1.184	0.184	0.121	LYY7-5
PB 588	HS6 R THIGH, CONTINUOUS S	951014	11.35	HS6 R THI	0.180	1.000	0.643	0.286	0.134	LYY7-6
PB 588	HS1 R THIGH 1, PROXIMAL	951111	11.57	HS1 R THI1	0.330	0.727	0.750	0.375	0.276	LYY8-1
PB 588	HS2 R THIGH, DISTAL	951111	11.58	HS2 R THI2	0.190	0.737	0.500	0.357	0.184	LYY8-2
PB 588	HS3 L THIGH, DONOR, MATUR	951111	11.59	HS3 L THI	0.560	1.000	0.827	0.077	0.068	LYY8-3
PB 588	HS4 L THIGH,DONOR, STILL AC	951111	12.00	HS4 L THI	0.290	0.690	0.348	0.261	0.142	LYY8-4
PB 588	HS1 R THIGH, LATERAL, HS	951209	11.52	HS1 R THI	0.360	0.861	0.731	0.385	0.173	LYY9-1
PB 588	HS1i, R THIGH, LATERAL, INJE	951209	11.54	HS1i R THI	0.570	1.000	1.163	0.163	0.107	LYY9-1i
PB 588	HS2 R THIGH, DISTAL	951209	11.55	HS2 R THI	0.300	0.733	0.522	0.304	0.195	LYY9-2
PB 588	HS3 L THIGH, DONOR, MATUR	951209	11.56	HS3 L THI	0.610	1.000	1.109	0.109	0.069	LYY9-3
PB 588	HS4 L THIGH, DISTAL	951209	11.56	HS4 L THI	0.240	1.000	0.556	0.333	0.141	LYY9-4
PB 588	NS R THIGH, ADJACENT TO HS	951209	11.57	NS R THI	0.550	1.000	1.078	0.078	0.077	LYY9-NS
BS 555	HS1 R THIGH, AFTER INJECTIO	960106	12.09	HS1 R THI	0.220	1.000	1.158	0.158	0.091	LYY10-1
PB 588	HS2 R THIGH, SSG	960106	12.10	HS2 R THI	0.390	0.897	0.765	0.147	0.094	LYY10-2
PB 588	HS3 L THIGH, DONOR	960106	12.12	HS3 L THI	0.550	0.927	0.904	0.058	0.051	LYY10-3D
PB 588	HS4 L THIGH	960106	12.13	HS4 L THI	0.210	0.762	0.500	0.312	0.177	LYY10-4D
PB 588	NS R THIGH	960106	12.14	NS R THI	0.480	0.958	0.956	0.067	0.071	LYY10-N1
PB 588	HS1 R THI	960203	12.11	HS1 R THI	0.490	0.633	0.564	0.256	0.116	LYY11-1
PB 588	HS2 R THIGH,DISTAL,SSG	960203	12.11	HS2	0.410	0.707	0.594	0.281	0.157	LYY11-2
PB 588	HS5 R THIGH,INJ	960203	12.12	HS5 INJ	0.440	1.000	1.189	0.189	0.158	LYY11-2
PB 588	HS5 R THIGH,INJ	960203	12.12	HS5 INJ	0.440	1.000	1.189	0.189	0.158	LYY11-5i
PB 588	HS3 L THIGH	960203	12.13	HS3 L THI	0.440	0.545	0.333	0.222	0.131	LYY11-3
PB 588	HS4 L THIGH,DISTAL	960203	12.14	HS4	0.380	0.553	0.310	0.310	0.147	LYY11-4
PB 588	NS R THIGH,MEDIAL	960203	12.14	NS	0.570	0.825	0.900	0.140	0.096	LYY11-N
PB 588	HS1 R THIGH 1,SSG	960302	12.19	HS1 R THI1	0.340	1.000	1.214	0.214	0.082	LYY12-1
PB 588	HS2 R THIGH,DISTAL	960302	12.20	HS2 R THI	0.390	0.641	0.500	0.300	0.127	LYY12-2
PB 588	HS3 MATURE DONOR, L THI	960302	12.21	HS3 L THI	0.600	1.000	0.982	0.091	0.060	LYY12-3
PB 588	HS INJECION SITE, R THIGH	960302	12.21	HS R THI i	0.560	1.000	0.938	0.167	0.110	LYY12-i
PB 588	HS4 L THIGH,DONOR	960302	12.22	HS4 L THI	0.240	1.000	1.600	0.600	0.254	LYY12-4
PB 588	NS R THIGH,MEDIAL	960302	12.23	NS1 R THI	0.530	1.000	1.104	0.104	0.080	LYY12-N
PB 588	HS1 R THIGH DISTAL	960330	11.29	HS1 R THI	0.280	0.714	0.591	0.273	0.112	LYY13-1
PB 588	HS2 R THIGH SSG MATURE	960330	11.30	HS2 R SSG	0.300	0.700	0.783	0.304	0.192	LYY13-2
PB 588	HS3 R THIGH, SCAR BAND	960330	11.30	HS3 R THI	0.270	0.704	0.667	0.286	0.155	LYY13-3
PB 588	HS4 R THIGH, INJECTION SITE	960330	11.31	HS4 INJ	0.340	0.765	0.714	0.214	0.143	LYY13-4i
PB 588	HS5 L THIGH,DISTAL, DONOR	960330	11.32	HS5 L THI	0.260	0.654	0.579	0.368	0.202	LYY13-5D
PB 588	HS6 L THIGH,PROXIMAL, DON	960330	11.33	HS6 L THI	0.360	0.917	0.968	0.161	0.102	LYY13-6D
PB 588	NS1 R THIGH, PROXIMAL	960330	11.33	NS1 R THI	0.380	0.895	1.000	0.187	0.111	LYY13-N1
PB 588	NS2 R THIGH, DISTAL	960330	11.34	NS2 R THI	0.290	0.931	1.130	0.261	0.170	LYY13-N2

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NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
BO 684	HS1 L THIGH	950615	17.08	HS1 L THI	0.190	0.895	1.000	0.357	0.201	PFL1-1
BO 684	NS R THIGH	950615	17.09	NS R THI	0.590	0.983	1.037	0.093	0.054	PFL1-1NS
BO 684	HS2 R KNEE, MEDIAL	950615	17.11	HS2 R KNEE	0.200	0.900	0.929	0.429	0.266	PFL1-2
BO 684	HS3 R LEG, 2/12 POST-INJURY	950615	17.11	HS3 R LEG	0.150	0.800	0.818	0.364	0.281	PFL1-3
BO 684	NS L LEG	950615	17.12	NS L LEG	0.320	0.969	1.037	0.185	0.138	PFL1-3NS
BO 684	HS1 R POPLITEAL	950727	16.50	HS1 R KNEE	0.220	0.773	0.765	0.294	0.164	PFL2-1
BO 684	HS2 R CALF	950727	16.51	HS2 R CALF	0.340	0.912	0.931	0.172	0.127	PFL2-2
BO 684	NS R THIGH	950727	16.52	NS R THIGH	0.500	0.980	0.957	0.087	0.067	PFL2-3NS
BO 684	HS3 L THIGH	950727	16.53	HS3 L THI	0.250	0.880	0.850	0.250	0.172	PFL2-3
BO 684	NS L CALF	950727	16.54	NS L CALF	0.320	0.906	0.963	0.185	0.156	PFL2-2NS
BO 684	NS1 R THIGH	950907	17.12	NS1 R THI	0.710	0.845	0.788	0.076	0.061	PFL3-1NS
BO 684	NS2 L KNEE	950907	17.13	NS2 L KNEE	0.630	0.841	0.810	0.086	0.072	PFL3-2NS
BO 684	NS3 L LEG	950907	17.14	NS3 L LEG	0.420	0.786	0.737	0.105	0.071	PFL3-3NS
BO 684	HS1 L THIGH	950907	17.15	HS1 L THI	0.270	1.000	0.714	0.286	0.203	PFL3-1
BO 684	HS2 R KNEE	950907	17.15	HS2 R KNEE	0.170	0.765	0.750	0.417	0.184	PFL3-2
BO 684	HS3 R LEG	950907	17.16	HS3 R LEG	0.320	0.813	0.680	0.280	0.159	PFL3-3
BO 684	HS1 L THIGH	951014	12.34	HS1 L THI	0.280	0.821	0.783	0.217	0.205	PFL4-1
BO 684	HS2 R KNEE	951014	12.35	HS2 R KNEE	0.270	0.815	0.714	0.286	0.189	PFL4-2
BO 684	HS3 R LEG	951014	12.35	HS3 R LEG	0.380	1.000	0.676	0.118	0.076	PFL4-2
BO 684	HS3 R LEG	951014	12.35	HS3 R LEG	0.380	1.000	0.676	0.118	0.076	PFL4-3
BO 684	HS1 L THIGH	951102	15.06	HS1 L THI	0.320	0.813	0.667	0.185	0.138	PFL5-1
BO 684	HS2 R KNEE	951102	15.07	HS2 R KNEE	0.260	0.769	0.667	0.238	0.162	PFL5-2
BO 684	HS2 R KNEE,	951102	15.07	HS2 R KNEE	0.260	0.769	0.667	0.238	0.162	PFL5-2
BO 684	HS3 R LEG	951102	15.08	HS3 R LEG	0.350	0.657	0.548	0.129	0.080	PFL5-3
BO 684	NS1 R THIGH	951102	15.09	NS1 R THI	0.730	1.000	1.159	0.159	0.101	PFL5-1N
BO 684	NS2 L LEG	951102	15.09	NS2 L LEG	0.440	0.977	0.756	0.073	0.046	PFL5-2N
BO 684	HS1 R KNEE, MEDIAL	951130	16.33	HS1 R KNEE	0.320	0.813	0.720	0.280	0.196	PFL6-1
BO 684	HS2 R CALF	951130	16.35	HS2 R CALF	0.300	1.000	0.708	0.250	0.175	PFL6-2
BO 684	HS3 L THIGH, DISTAL	951130	16.36	S3 L THIGH	0.250	0.840	0.800	0.250	0.182	PFL6-3
BO 684	HS1 L THIGH	951228	17.40	HS1 L THI	0.250	0.840	0.750	0.250	0.197	PFL7-1
BO 684	HS2 R KNEE	951228	17.40	HS2 R KNEE	0.150	0.933	0.700	0.500	0.202	PFL7-2
BO 684	HS3 R LEG	951228	17.42	HS R R LEG	0.240	1.000	1.333	0.333	0.198	PFL7-3
BO 684	NS1 R THIGH	951228	17.42	NS1 R THI	0.480	1.000	1.091	0.091	0.073	PFL7-N1
BO 684	NS2 L KNEE, MEDIAL	951228	17.43	NS2 L KNEE	0.620	0.935	0.786	0.107	0.082	PFL7-N2
BO 684	NS3 L LEG	951228	17.44	NS3 R LEG	0.430	0.767	0.725	0.075	0.045	PFL7-N3
BO 684	HS1 L THIGH	960125	17.21	HS1 L THI	0.350	0.600	0.607	0.250	0.176	PFL8-1
BO 684	HS2 R THIGH, MEDIAL	960125	17.22	HS2 R THI	0.640	1.000	1.208	0.208	0.120	PFL8-2
BO 684	HS3 R LEG	960125	17.22	HS3 R LEG	0.250	1.000	1.316	0.316	0.174	PFL8-3
BO 684	NS1 R THIGH	960125	17.24	NS1 R THI	0.530	1.000	1.178	0.178	0.104	PFL8-N1
BO 684	NS2 L KNEE	960125	17.24	NS2 L KNEE	0.640	1.000	0.893	0.143	0.107	PFL8-N2
BO 684	NS3 L LEG	960125	17.25	NS2 L KNEE	0.420	1.000	0.946	0.135	0.103	PFL8-N3
BO 684	HS1 L THIGH	960302	11.48	HS1 L THI	0.330	0.576	0.500	0.269	0.163	PFL9-1
BO 684	HS2 R KNEE	960302	11.48	HS2 R KNEE	0.390	0.615	0.600	0.300	0.178	PFL9-2
BO 684	HS3 R LEG	960302	11.49	HS3 R LEG	0.310	1.000	0.913	0.348	0.219	PFL9-3
BO 684	NS1 R THIGH	960302	11.50	NS1 R THI	0.580	1.000	1.094	0.094	0.064	PFL9-N1
BO 684	NS2 L KNEE	960302	11.51	NS2 L KNEE	0.670	0.821	0.763	0.136	0.092	PFL9-N2
BO 684	NS3 L LEG	960302	11.51	NS3 L LEG	0.380	1.000	1.152	0.152	0.106	PFL9-N3
BO 684	HS1 R KNEE	960411	17.29	HS1 R KNEE	0.100	0.700	0.714	0.429	0.211	PFL10-1
BO 684	HS2 R LEG	960411	17.30	HS2 R LEG	0.210	0.857	0.765	0.235	0.164	PFL10-2
BO 684	HS3 L THIGH,	960411	17.31	HS3 L THI	0.160	0.813	0.769	0.231	0.139	PFL10-3
BO 684	NS1 R THIGH	960411	17.32	NS1 R THI	0.400	0.950	1.000	0.111	0.078	PFL10-N1
BO 684	NS2 L KNEE	960411	17.33	NS2 L KNEE	0.310	0.935	0.964	0.107	0.052	PFL10-N2
BO 684	NS3 L LEG	960411	17.33	NS3 L LEG	0.360	0.972	0.970	0.091	0.089	PFL10-N3
BO 684	HS1 L THIGH DISTAL	960509	17.10	HS1 L THI	0.220	0.909	0.842	0.158	0.105	PFL11-1
BO 684	HS2 L THIGH PROXIMAL	960509	17.11	HS2 L THI	0.180	0.778	0.714	0.286	0.113	PFL11-2
BO 684	HS3 R CALF	960509	17.12	HS3 R CALF	0.260	0.692	0.619	0.238	0.164	PFL11-3
BO 684	HS4 R THIGH	960509	17.13	HS4 R THI	0.290	0.793	0.783	0.261	0.135	PFL11-4
BO 684	NS1 R THIGH	960509	17.13	N1 R THI	0.580	0.966	0.906	0.094	0.057	PFL11-N1
BO 684	NS2 L CALF	960509	17.14	N2 L CALF	0.400	0.925	0.917	0.111	0.091	PFL11-N2
BO 684	HS1 L THIGH PROXIMAL	960627	17.20	HS1 L THI	0.000	0.000	0.000	0.000	0.000	PFL12-1
BO 684	HS2 L THIGH DISTAL	960627	17.21	HS2	0.370	0.676	0.645	0.194	0.112	PFL12-2
BO 684	HS3 R KNEE	960627	17.21	HS2	0.250	0.520	0.500	0.250	0.120	PFL12-3
BO 684	HS4 R CALF	960627	17.22	HS4 R CALF	0.290	0.586	0.440	0.160	0.109	PFL12-4
BO 684	NS R THIGH	960627	17.23	NS R THI	0.470	0.872	0.951	0.146	0.078	PFL12-N1
BO 684	NS2 L KNEE	960627	17.24	NS L KNEE	0.550	0.891	0.878	0.122	0.072	PFL12-N2
BO 684	NS3 L CALF	960627	17.24	NS L CALF	0.430	0.837	0.865	0.162	0.105	PFL12-N3

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PS 318	HS1 R BUTTOCK	950518	15.27	HS1 R BUT	0.200	0.900	0.933	0.333	0.189	SNF1-1
PS 318	HS2 MIDDLE OF BUTTOCK	950518	15.29	HS2 M BUT	0.210	0.905	0.938	0.312	0.148	SNF1-2
PS 318	HS3 L BUTTOCK	950518	15.30	HS3 L BUT	0.170	0.824	0.917	0.417	0.290	SNF1-3
PS 318	NS BELOW R BUTTOCK	950518	15.31	NS R BUT	0.400	0.975	1.029	0.143	0.088	SNF1-4NS
PS 318	HS1 L BACK	950615	16.06	HS1 L BACK	0.170	0.824	0.769	0.308	0.231	SNF2-1
PS 318	HS2 BACK, MIDDLE	950615	16.07	HS2 ME BAC	0.350	0.886	0.931	0.207	0.144	SNF2-2
PS 318	HS3 R BACK	950615	16.09	HS2 R BACK	0.180	0.833	0.833	0.500	0.248	SNF2-3
PS 318	NS R BUTTOCK JUST BELOW	950615	16.09	NS R BUT	0.500	0.960	1.049	0.220	0.152	SNF2-3NS
PB 612	HS1 R BUTTOCK	950713	15.49	HS1 BACK R	0.200	0.700	0.437	0.250	0.175	SNF3-1
PB 612	HS2 MID-BUTTOCK, MATURE	950713	15.50	HS2 M BACK	0.210	0.857	0.765	0.235	0.105	SNF3-2
PB 612	HS3 L BUTTOCK	950713	15.53	HS3 L BACK	0.160	1.000	0.615	0.231	0.209	SNF3-3
PS 318	NS R BUTTOCK	950713	15.54	NS L BUTT	0.490	0.939	1.023	0.140	0.101	SNF3-3NS
PS 318	HS1 L BACK, STILL ACTIVE, 5/1	950727	14.30	HS1 L BACK	0.280	0.893	0.870	0.217	0.110	SNF4-1
PS 318	HS2 R BACK	950727	14.31	HS2 R BACK	0.210	0.714	0.529	0.235	0.146	SNF4-2
PS 318	HS3 MID-BACK, FLAT AND SOF	950727	14.32	HS3 M BACK	0.340	0.794	0.862	0.172	0.082	SNF4-3
PS 318	NS R BUTTOCK, BELOW HS3 R	950727	14.33	NS R BUTT	0.390	1.000	1.059	0.147	0.106	SNF4-NS
PS 318	HS1 R BUTTOCK, ACTIVE, FIR	950921	15.36	HS1 R BUT	0.270	0.778	0.522	0.174	0.147	SNF5-1
PS 318	HS2 MID-BUTTOCK, MATURE	950921	15.37	HS2 MID-BU	0.250	0.800	0.800	0.250	0.138	SNF5-2
PS 318	HS3 L BUTTOCK, FIRM	950921	15.38	HS3 L BUT	0.140	0.857	0.545	0.273	0.171	SNF5-3
PS 318	NS R BUTTOCK	950921	15.39	NS R BUT	0.460	0.957	0.976	0.095	0.049	SNF5-1NS
PS 318	HS1 R BUTTOCK, PT MOVES A	951014	11.19	HS1 R BUT	0.260	0.808	0.619	0.238	0.169	SNF7-1
PS 318	HS2 MED BUTTOCK	951014	11.21	HS2 M BUT	0.380	1.000	0.727	0.152	0.086	SNF7-2
PS 318	HS3 L BUTTOCK	951014	11.21	HS3 L BUT	0.240	0.792	0.579	0.263	0.182	SNF7-3
PS 318	HS1 R BUTTOCK	951111	10.19	HS1 R BUT	0.220	0.727	0.500	0.222	0.122	SNF8-1
PS 318	HS2 MID-BUTTOCK	951111	10.20	HS2 M-BUT	0.660	0.667	0.661	0.119	0.056	SNF8-2
PS 318	HS3 L BUTTOCK	951111	10.21	HS3 L-BUT	0.250	0.680	0.450	0.250	0.134	SNF8-3
PS 318	HS1 R BUTTOCK	951209	11.00	HS1 R BUT	0.220	0.682	0.500	0.222	0.144	SNF9-1
PS 318	HS2 MID-BUTTOCK, MATURE	951209	11.01	HS2 M BUT	0.520	1.000	0.804	0.130	0.074	SNF9-2
PS 318	HS3 L BUTTOCK	951209	11.02	HS3 L BUT	0.180	0.722	0.500	0.500	0.350	SNF9-3
PS 318	NS, R BUTTOCK, JUST BELOW	951209	11.02	NS R BUT	0.630	0.952	0.895	0.105	0.069	SNF9-NS
PS 318	HS1 R BUTTOCK	960106	11.39	HS1 R BUT	0.180	0.778	0.615	0.385	0.241	SNF10-1
PS 318	HS2 MID-BUTTOCK	960106	11.40	HS2 M BUT	0.460	0.891	0.902	0.122	0.066	SNF10-2
PS 318	HS3 L BUTTOCK	960106	11.41	HS3 L BUT	0.340	0.647	0.467	0.133	0.082	SNF10-3
PS 318	NS R BUTTOCK	960106	11.42	NS1 R BUT	0.610	0.934	0.930	0.070	0.047	SNF10-NS
PS 318	HS1 R BUTTOCK, STOP PG 1/1	960302	10.20	HS1 R BUT	0.180	0.611	0.636	0.636	0.323	SNF11-1
PS 318	HS2 MID-BUTTOCK, MATURE	960302	10.22	HS3 R BUT	0.480	0.938	0.875	0.200	0.098	SNF11-2
PS 318	HS3 L BUT	960302	10.24	HS3 L BUT	0.400	0.525	0.300	0.333	0.167	SNF11-3
PS 318	NS R BUTTOCK	960302	10.25	NS R BUT	0.620	1.000	0.926	0.148	0.100	SNF11-NS
PS 318	HS1 BACK R, STILL ACTIVE	960330	11.36	HS1 BACK R	0.300	0.600	0.458	0.250	0.171	SNF12-1
PS 318	HS2 MATURE BACK, MID	960330	11.37	HS2 BACK M	0.320	0.844	0.920	0.280	0.160	SNF12-2
PS 318	HS3 L BACK	960330	11.37	HS3 L BACK	0.450	0.711	0.647	0.324	0.176	SNF12-3
PS 318	HS3 L BACK	960330	11.37	HS3 L BACK	0.450	0.711	0.647	0.324	0.176	SNF12-3
PS 318	NS R BACK	960330	11.39	NS R BACK	0.390	0.872	0.970	0.182	0.088	SNF12-N
PS 318	NORMAL SKIN	960518	10.12	NS R BUT	0.480	0.979	1.000	0.091	0.055	SNF13-N
PS 318	HS1 L BACK	960518	10.13	HS1 L BACK	0.200	0.900	0.800	0.333	0.228	SNF13-1
PS 318	HS2 MID-BACK, MATURE	960518	10.14	HS2 MID	0.270	0.926	0.917	0.125	0.070	SNF13-2
PS 318	HS3 R BACK	960518	10.15	HS3 R BACK	0.190	0.842	0.600	0.267	0.188	SNF13-3
PS 318	HS1,R BUTTOCK,ACTIVE	960622	9.58	HS1 R BUT	0.200	0.650	0.563	0.250	0.129	SNF14-1
PS 318	HS2 MID-BUTTOCK,MATURE	960622	9.59	HS2 M BUT	0.330	0.909	0.931	0.138	0.120	SNF14-2
PS 318	HS3 L BUTTOCK	960622	10.00	HS3 L BUT	0.290	0.724	0.609	0.261	0.143	SNF14-3
PS 318	NS R BUTTOCK	960622	10.01	NS R BUT	0.460	0.891	1.000	0.179	0.122	SNF14-N1

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 626	HS2, R ANKLE, ACTIVE	1/4/95	10.15	HS2 R ANK	0.050	1.000	1.000	0.667	0.262	SWY2
PB 626	HS1 L ANKLE ABOVE MALLEOL	1/4/95	10.16	HS1 L ANK	0.080	0.750	1.000	1.000	0.486	SWY1
PB 626	HS3 R BACK, ACTIVE	1/4/95	10.18	HS3 R BACK	0.090	0.889	1.000	0.500	0.327	SWY3
PB 626	HS4 L THIGH, POSTERIOR	1/4/95	10.19	HS4 L THI	0.210	0.905	0.867	0.400	0.251	SWY4
PB 626	NS R THIGH, POSTERIOR	1/4/95	10.21	NS R THI	0.510	0.941	0.913	0.109	0.033	SWY5-NS
PB 626	NS L BACK	1/4/95	10.21	NS L BACK	0.450	0.933	1.077	0.154	0.084	SWY6-NS
PB 626	L MEDIAL MALLEOLUS, FLAT P	950302	16.06	L MED MALL	0.110	0.909	0.750	0.375	0.180	SWY3-1
PB 626	R ANKLE, ACTIVE, FIRM SCAR	950302	16.08	R ANKLE	0.090	0.667	0.800	0.800	0.318	SWY3-2
PB 626	R SIDE OF SCAR AT BACK, RAI	950302	16.11	R BACK	0.190	0.895	0.769	0.462	0.277	SWY3-3
PB 626	POSTERIAL L THIGH, RAISED,	950302	16.12	L THIGH	0.120	0.917	0.778	0.333	0.248	SWY3-4
PB 626	HS3, R BACK, ACTIVE	950504	16.43	HS3 R BACK	0.160	0.750	0.727	0.455	0.283	SWY4-3
PB 626	HS4 L THIGH POSTERIOR	950504	16.46	HS4 L THI	0.110	0.909	0.750	0.375	0.203	SWY4-3
PB 626	HS4 L THIGH, POSTERIOR	950504	16.46	HS4 L THI	0.110	0.909	0.750	0.375	0.203	SWY4-4
PB 626	HS1 R ANKLE	950504	16.48	HS1 R ANK	0.110	0.818	0.625	0.375	0.231	SWY4-1
PB 626	HS2, L ANKLE ABOVE MALLEO	950504	16.49	HS2 L ANK	0.200	0.950	0.800	0.333	0.203	SWY4-2
PB 626	NS L BACK VS SWY4-3	950504	16.50	HS2 L ANK	0.750	0.960	1.061	0.136	0.102	SWY4-3N
PB 626	NS POSTERIOR L BACK	950504	16.52	NS R THI P	0.760	0.961	0.986	0.086	0.071	SWY4-4
PB 626	NS R THIGH, VS SWY4-4	950504	16.52	NS R THI P	0.760	0.961	0.986	0.086	0.071	SWY4-4N
PB 626	HS1 L MEDIAL MALLEOLUS	950601	15.35	HS1 L MAL	0.150	0.933	0.818	0.364	0.177	SWY5-1
PB 626	HS2 R ANKLE, MOVING	950601	15.37	HS2 R ANK	0.150	0.467	0.429	0.071	0.101	SWY5-2
PB 626	HS3 R BACK	950601	15.38	HS3 R BACK	0.230	0.652	0.643	0.643	0.330	SWY5-3
PB 626	HS4 R THIGH, POSTERIOR, NO	950601	15.38	HS4 R THI	0.170	0.706	0.769	0.308	0.174	SWY5-4
PB 626	NS L THIGH	950601	15.39	NS L THI	0.630	0.968	0.983	0.086	0.075	SWY5-4NS
PB 626	NS L BACK	950601	15.40	NS L BACK	0.540	0.963	1.021	0.125	0.098	SWY5-3NS
PB 626	HS1 BACK	950701	12.51	HS1 BACK	0.110	1.000	0.714	0.571	0.389	SWY6-1
PB 626	HS2 R BACK	950701	12.52	HS2 R BACK	0.140	0.714	0.889	0.556	0.247	SWY6-2
PB 626	NS1 L BACK	950701	12.53	NS1 L BACK	0.770	0.948	0.986	0.085	0.053	SWY6-1NS
PB 626	NS2 R POSTERIOR THIGH	950701	12.54	NS2 R THI	0.520	1.000	1.067	0.156	0.102	SWY6-3NS
PB 626	HS3 L POSTERIOR THIGH	950701	12.55	HS3 L THI	0.140	0.714	0.667	0.556	0.219	SWY6-3
PB 626	HS4 L LATERAL MALLEOLUS	950701	12.56	HS4 L ANK	0.070	0.857	1.000	0.750	0.359	SWY6-4
PB 626	HS5 L ANKLE, NEAR MEDIAL M	950701	12.57	HS5 R ANK	0.090	0.778	0.833	0.500	0.479	SWY6-5
PB 626	HS1 R ANKLE, MEDIAL MALLE	950727	15.32	HS1 R ANK	0.260	0.769	0.579	0.368	0.213	SWY7-1
PB 626	HS2 R ANKLE, ABOVE TA	950727	15.33	HS2 R TA	0.130	0.692	0.556	0.444	0.200	SWY7-2
PB 626	HS3 L ANKLE, LATERAL MALLE	950727	15.34	HS3 L ANK	0.100	0.900	0.429	0.429	0.176	SWY7-3
PB 626	HS4 L THIGH, POSTERIOR	950727	15.35	HS4 L THI	0.090	0.889	0.833	0.500	0.217	SWY7-4
PB 626	HS5 L BACK	950727	15.36	HS5 L BACK	0.280	0.714	0.565	0.217	0.152	SWY7-5
PB 626	HS6 R BACK	950727	15.37	HS6 R BACK	0.230	0.870	0.579	0.211	0.123	SWY7-6
PB 626	HS1 L MEDIAL MALLEOLUS	950907	16.22	HS1	0.160	0.813	0.667	0.333	0.175	SWY8-1
PB 626	HS2 R ANKLE	950907	16.23	HS2	0.210	1.000	1.167	0.167	0.119	SWY8-2
PB 626	HS2 R ANLKE	950907	16.23	HS2	0.210	1.000	1.167	0.167	0.119	SWY8-3
PB 626	HS3 BACK	950907	16.24	HS3	0.150	0.733	0.778	0.667	0.285	SWY8-3
PB 626	HS4 L THIGH, BACK	950907	16.25	HS4	0.150	1.000	0.769	0.154	0.046	SWY8-4
PB 626	HS1 L ANKLE, MEDIAL MALLEO	951005	16.56	HS1 L ANK	0.360	0.583	0.323	0.161	0.105	SWY9-1
PB 626	HS2 R TA	951005	16.57	HS2 R TA	0.260	1.000	0.571	0.238	0.121	SWY9-2
PB 626	HS3 BACK	951005	16.58	HS3 BACK	0.230	0.739	0.900	0.150	0.076	SWY9-3
PB 626	HS4 L THIGH	951005	16.59	HS4 L THI	0.250	0.680	0.429	0.190	0.113	SWY9-4
PB 626	HS1 L MEDIAL MALLEOLUS	951102	14.04	HS1 L ANK	0.230	0.826	0.556	0.278	0.165	SWY10-1
PB 626	HS2 R ANKLE	951102	14.05	HS2 R ANK	0.190	0.789	0.571	0.357	0.172	SWY10-2
PB 626	HS3 R BACK	951102	14.06	HS3 BACK	0.130	0.923	1.000	0.300	0.232	SWY10-3
PB 626	HS4 L THIGH	951102	14.07	HS4 L THI	0.340	0.706	0.552	0.172	0.090	SWY10-4
PB 626	NS1 L BACK	951102	14.08	NS1 BACK	0.630	0.714	0.655	0.086	0.064	SWY10-1N
PB 626	NS2 R THIGH	951102	14.09	NS2 R THI	0.450	1.000	1.100	0.125	0.084	SWY10-2N
PB 626	HS1 L ANKLE, MEDIAL MALLEO	951214	16.47	HS1 L ANK	0.270	0.593	0.429	0.286	0.229	SWY10-1
PB 626	HS1 L ANKLE, MEDIAL MALLEO	951214	16.47	HS1 L ANK	0.270	0.593	0.429	0.286	0.229	SWY11-1
PB 626	HS1 L ANKLE, MEDIAL MALLEO	951214	16.47	HS1 L ANK	0.270	0.593	0.429	0.286	0.229	SWY9-1
PB 626	HS2 R ANKLE, TA	951214	16.50	HS2 R ANK	0.180	1.000	1.385	0.385	0.284	SWY11-2
PB 626	HS3 R BACK	951214	16.51	HS3 R BACK	0.140	1.000	1.000	0.400	0.174	SWY11-3
PB 626	HS4 L THIGH, POSTERIOR	951214	16.52	HS4 L THI	0.160	1.000	0.818	0.455	0.250	SWY11-4
PB 626	HS4 POSTERIOR L THIGH	951214	16.53	HS4 L THI	0.330	0.848	0.643	0.179	0.104	SWY10-4B
PB 626	HS3 R BACK	960111	15.23	HS3 R BACK	0.340	0.735	0.480	0.360	0.233	SWY12-3
PB 626	NS3 L BACK	960111	15.24	NS3 L BACK	0.530	0.925	0.913	0.152	0.092	SWY12-3N
PB 626	HS1 L MEDIAL MALLEOLUS	960111	15.26	HS1 L ANK	0.380	0.737	0.250	0.187	0.106	SWY12-1
PB 626	NS1 L ANKLE, ANTERIOR TO M	960111	15.27	NS1 L ANK	0.520	0.712	0.533	0.156	0.085	SWY12-1N
PB 626	HS2 R TA	960111	15.28	HS2 R TA	0.220	0.727	0.437	0.375	0.219	SWY12-2
PB 626	HS4 L THIGH, POST	960111	15.29	HS4 L THI	0.190	0.842	0.625	0.187	0.157	SWY12-4
PB 626	NS4 L THIGH, ADJACENT TO H	960111	15.29	NS4 L THI	0.660	1.000	1.050	0.100	0.064	SWY12-4
PB 626	NS4 L THIGH, ADJ TO HS4	960111	15.29	NS4 L THI	0.660	1.000	1.050	0.100	0.064	SWY12-4N
PB 626	HS1 L MEDIAL MALLEOLUS	960203	12.27	HS1 L ANK	0.260	0.385	0.278	0.444	0.165	SWY13-1
PB 626	HS2 R ANKLE	960203	12.28	HS2 R ANK	0.180	1.000	0.500	0.286	0.110	SWY13-2
PB 626	HS3 R BACK	960203	12.30	HS3 BACK	0.430	0.651	0.543	0.229	0.143	SWY13-3
PB 626	NS1 R BACK	960203	12.30	NS1 BACK	0.830	0.843	0.867	0.107	0.078	SWY13-N3
PB 626	HS4 L THIGH,POSTERIOR	960203	12.31	HS4 L THI	0.410	0.610	0.514	0.171	0.100	SWY13-4
PB 626	NS2 R THIGH,POSTERIOR	960203	12.32	NS R THI	0.660	0.848	0.877	0.158	0.097	SWY13-N4

[illegible]

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
BS 570	HS4 L THIGH, APPROACH MAT	3/11/95	11.44	HS4 L THI	0.220	0.864	0.722	0.222	0.128	TSC2-4
BS 570	HS4 L THIGH, NEAR MATURE,	3/11/95	11.45	HS4 L THI	0.200	0.850	0.688	0.250	0.142	TSC2-4B
BS 570	PUBIC HS, RAISED, FIRM, ITCH	950311	11.31	PUBIC/HS	0.190	0.947	0.933	0.267	0.186	TSC2-1A
BS 570	PUBIC HS, RAISED, FIRM, 2ND	950311	11.34	PUBIC/HS	0.170	0.765	0.769	0.308	0.220	TSC2-1B
BS 570	L THIGH, HS ON SSG, RAISED,	950311	11.37	L THIGH/HS	0.150	0.933	0.833	0.250	0.116	TSC2-2
BS 570	L THIGH HS ON SSG, 2ND MEA	950311	11.38	L THIGH/HS	0.120	0.917	0.889	0.333	0.156	TSC2-2B
BS 570	L THIGH, MEDIAL NS, VS TSC2-	950311	11.40	L THIGH/NS	0.490	0.918	0.976	0.167	0.121	TSC2-7
BS 570	GROIN, HS1, INJECTION 3/52	950413	17.15	GROIN HS1	0.250	0.840	0.789	0.316	0.158	TSC3-1
BS 570	GROIN, NS	950413	17.17	GROIN NS	0.700	0.971	0.969	0.094	0.078	TSC3-1N
BS 570	L THIGH, SSG, ACTIVE HS2	950413	17.18	L THI, HS2	0.220	0.864	0.824	0.294	0.140	TSC3-2
BS 570	R THIGH, SSG, MATURE HS3	950413	17.19	R THI, SSG	0.480	0.958	1.000	0.116	0.073	TSC3-3
BS 570	L THIGH, NS	950413	17.20	L THIGH NS	0.430	0.953	1.026	0.132	0.089	TSC3-23N
BS 570	L THIGH ,DONAR	950413	17.22	L THIGH D	0.320	0.906	0.885	0.231	0.159	TSC3-5
BS 570	R THIGH, NS	950413	17.23	R THI, NS	0.270	0.963	1.043	0.174	0.090	TSC3-5N
BS 570	HS1 L GROIN	950504	17.02	HS1 L GRON	0.260	0.846	0.789	0.368	0.227	TSC4-1
BS 570	NS R GROIN, VS TSC4-1	950504	17.04	NS R GRON	0.640	0.969	0.983	0.085	0.059	TSC4-1N
BS 570	HS2, L THIGH	950504	17.05	HS2 L THI	0.140	0.857	0.800	0.400	0.171	TSC4-2
BS 570	HS3 R THIGH, MATURE SSG	950504	17.06	HS3 R THI	0.460	0.957	1.050	0.150	0.109	TSC4-3
BS 570	HS5 L THIGH, MATURE DONAR	950504	17.07	HS4 L THI	0.340	0.912	0.926	0.259	0.160	TSC4-5
BS 570	NS L THIGH, VS HS2,4 & 5	950504	17.07	NS L THI	0.420	0.952	1.054	0.135	0.088	TSC4-5N
BS 570	HS1 L GROIN	950610	10.53	HS1 L GRON	0.190	0.842	0.786	0.357	0.180	TSC5-1
BS 570	HS1 L GROIN, 2ND MX	950610	10.54	HS1 L GRON	0.260	0.846	0.789	0.368	0.209	TSC5-1B
BS 570	HS1 L GROIN, 3RD MX	950610	10.55	HS1 L GRON	0.240	0.875	0.789	0.263	0.144	TSC5-1C
BS 570	HS2 L THIGH, SSG	950610	10.55	HS2 L THI	0.220	0.909	0.889	0.222	0.109	TSC5-2
BS 570	HS3 R THIGH, SSG	950610	10.57	HS3 R THI	0.530	0.943	1.000	0.152	0.103	TSC5-3
BS 570	NS R GROIN	950610	10.58	NS R GROIN	0.630	0.984	1.000	0.068	0.051	TSC5-1NS
BS 570	HS3 R THIGH, SSG, 2ND MX	950610	10.58	HS3 R THI	0.500	1.000	1.163	0.163	0.088	TSC5-3B
BS 570	NS L THIGH	950610	10.59	NS L THI	0.500	0.980	1.091	0.136	0.091	TSC5-2NS
BS 570	HS4 L THIGH DONOR	950610	11.00	D L THIGH	0.350	0.943	0.933	0.167	0.101	TSC5-4
BS 570	HS1 GROIN, SOFTER	950727	15.14	HS1 GROIN	0.340	0.853	0.679	0.214	0.148	TSC6-1
BS 570	NS1 L GROIN,	950727	15.15	NS1 GROIN	0.690	0.957	0.954	0.062	0.043	TSC6-1NS
BS 570	HS2 L THIGH, SSG	950727	15.16	HS2 L THI	0.320	0.906	0.926	0.185	0.149	TSC6-2
BS 570	HS2 R THIGH, SSG	950727	15.17	HS3 R THI	0.540	0.981	1.109	0.174	0.112	TSC6-3
BS 570	HS4 L THIGH, DONAR	950727	15.18	HS4 L THI	0.400	0.900	0.771	0.143	0.077	TSC6-4

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 190	NS L ELBOW, VS TSK2-2	4/8/95	10.48	NS L ELBOW	0.420	0.952	1.057	0.200	0.131	TSK2-2N
PB 190	NS L FOREARM, VS TSK2-3	4/8/95	10.49	NS L FOREA	0.400	0.950	1.029	0.176	0.136	TSK2-3N
PB 190	R ARM, HS1, PINKISH, MILDLY	950408	10.36	R ARM HS1	0.220	0.773	0.765	0.294	0.188	TSK2-1
PB 190	R ELBOW, MATURE SG, NOT A	950408	10.39	R ELBOW S	0.220	0.909	0.882	0.294	0.147	TSK2-2
PB 190	R FOREARM HS3, STILL ACTIV	950408	10.41	R FOREARM	0.140	0.643	0.455	0.273	0.154	TSK2-3
PB 190	R FOREARM HS3, STILL ACTIV	950408	10.43	R FOREARM	0.150	0.800	0.583	0.250	0.123	TSK2-3B
PB 190	R FOREARM, HS3, 3RD MX	950408	10.44	R FOREARM	0.150	0.800	0.545	0.364	0.199	TSK2-3C
PB 190	L ARM, NS, VS TSK2-1	950408	10.45	L ARM, NS	0.580	0.966	1.059	0.137	0.100	TSK2-1N
PB 19	HS1 R ARM	950610	11.19	HS1 R ARM	0.140	0.857	0.727	0.273	0.244	TSK4-1
PB 19	HS1 R ARM, 2ND MX	950610	11.20	HS1 R ARM	0.200	0.900	0.824	0.176	0.092	TSK4-1B
PB 19	HS2 R CUBITAL FOSSA,SSG	950610	11.21	HS2 R ELB	0.240	0.958	1.000	0.143	0.105	TSK4-2
PB 19	HS3 R FOREARM,	950610	11.22	HS3 R FOR	0.230	0.826	0.778	0.278	0.193	TSK4-3
PB 19	NS L ARM	950610	11.23	NS R ARM	0.370	0.973	1.065	0.194	0.106	TSK4-1NS
PB 19	NS L CUBITAL FOSSA	950610	11.24	NS R ELB	0.450	0.978	1.050	0.125	0.084	TSK4-2NS
PB 19	NS L FOREARM	950610	11.24	NS L FOREA	0.420	0.952	1.028	0.167	0.115	TSK4-3NS
PB 190	HS1 R ARM	950810	15.32	HS1 R ARM	0.200	0.950	0.882	0.176	0.088	TSK5-1
PB 190	HS1 R ARM, 2ND MX	950810	15.32	HS1 R ARM	0.300	0.867	0.739	0.304	0.138	TSK5-1B
PB 190	HS2 R ELBOW	950810	15.33	HS1 R ARM	0.290	0.897	0.783	0.261	0.188	TSK5-2
PB 190	HS2 R ELBOW	950810	15.34	HS2 R ELB	0.280	1.000	0.826	0.217	0.183	TSK5-2B
PB 190	HS3 R FOREARM, 1/12 AFTER I	950810	15.34	HS3 R FORA	0.270	0.852	0.708	0.125	0.081	TSK5-3
PB 190	HS3 R FOREARM, 1/12 AFTER I	950810	15.36	HS3 R FORA	0.400	0.900	0.857	0.143	0.109	TSK5-3B
PB 190	HS4 R FOREARM	950810	15.37	HS3 R FORA	0.180	0.944	0.867	0.200	0.113	TSK5-4
PB 190	HS4 R FOREARM, 2ND MX	950810	15.38	HS4 R FORA	0.180	0.833	0.714	0.286	0.170	TSK5-4B
PB 190	NS1 R ARM	950810	15.40	NS1 L ARM	0.420	0.952	0.947	0.105	0.077	TSK5-1NS
PB 190	NS2 L ELBOW	950810	15.41	NS2 L ELB	0.470	0.915	0.975	0.175	0.116	TSK5-2NS
PB 190	NS3 L FOREARM	950810	15.42	NS3 L FORA	0.370	0.919	0.909	0.121	0.074	TSK5-3NS
PB 190	HS1 R ARM	950916	11.45	HS1 R ARM	0.200	0.900	0.625	0.250	0.121	TSK6-1
PB 190	HS2 LATERAL TO R CUBITAL F	950916	11.47	HS2 CUB FO	0.350	0.714	0.633	0.167	0.119	TSK6-2
PB 190	HS3 R FOREARM	950916	11.48	HS3 R FOR	0.280	1.000	1.167	0.167	0.122	TSK6-3
PB 190	HS4 R FOREARM, ATROPHIC S	950916	11.49	HS4 R FOR	0.310	0.968	0.923	0.192	0.129	TSK6-4
PB 190	NS1 L ARM	950916	11.50	NS1 L ARM	0.510	1.000	0.978	0.109	0.083	TSK6-1NS
PB 190	NS2 L LAT TO CUBITAL FOSSA	950916	11.51	NS2 L ELB	0.420	0.952	1.000	0.135	0.082	TSK6-2NS
PB 190	NS3 L FOREARM	950916	11.51	NS3 L FORA	0.460	0.935	0.929	0.095	0.063	TSK6-3NS
PB 190	HS1 R ARM	951014	12.08	HS1 R ARM	0.270	0.741	0.619	0.286	0.208	TSK7

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
BO 634	HS4 L WRIST, DORSUM, VS TS	3/11/95	12.18	HS4 L WRIS	0.110	0.727	0.714	0.571	0.287	TSY2-4B
BO 634	HS4 L WRIST DORSUM,VS TSY	3/11/95	12.19	HS4 L WRIS	0.070	0.714	0.800	0.400	0.160	TSY2-4C
BO 634	NS1 L WRIST VENTRAL	4/20/96	12.38	NS2 R DOR	0.430	0.907	0.868	0.132	0.089	TSY8-N1
BO 634	R WRIST VOLAR, HS, SOFTER	950311	12.10	R WRIST/V	0.170	0.882	0.769	0.308	0.211	TSY2-1
BO 634	R WRIST, DORSUM HS, FIRM	950311	12.11	R WRIST/D	0.190	0.895	0.786	0.357	0.241	TSY2-2
BO 634	L WRIST, VOLAR, HS	950311	12.13	L WRIST/V	0.310	0.774	0.538	0.192	0.105	TSY2-3
BO 634	L WRIST, DORSUM, HS	950311	12.13	L WRIST/D	0.310	0.774	0.538	0.192	0.105	TSY2-3A
BO 634	L WRIST DORSUM, HS, FIRM	950311	12.16	L WRIST/D	0.070	0.714	0.800	0.400	0.194	TSY2-4
BO 634	HS1, R WRIST VOLAR, 24MTH	950429	12.17	HS1 R WR V	0.190	0.789	0.857	0.357	0.280	TSY3-1
BO 634	HS2, R WRIST, DORSUM	950429	12.18	HS2 R WR D	0.170	0.824	0.833	0.417	0.267	TSY3-2
BO 634	L WRIST VOLAR, HS3	950429	12.20	HS3 L WR V	0.250	0.760	0.562	0.562	0.266	TSY3-3
BO 634	HS4 L WRIST DORSUM	950429	12.21	HS4 L WR D	0.140	0.714	0.667	0.556	0.348	TSY3-4
BO 634	HS4 L WRIST DORSUM	950429	12.21	HS4 L WR D	0.140	0.714	0.667	0.556	0.348	TSY3-4
BO 634	NS L WRIST DORSUM	950429	12.22	NS L WR D	0.450	0.911	0.895	0.184	0.114	TSY3-2N
BO 634	NS L WRIST VOLAR VS TSY3-1	950429	12.24	NS L WR V	0.610	0.934	0.904	0.173	0.128	TSY3-1N
BO 634	HS1 R WRIST VOLAR	950601	16.03	HS1 R W V	0.320	0.844	0.692	0.231	0.125	TSY4-1
BO 634	HS2 R WRIST DORSUM	950601	16.04	HS1 R W D	0.260	0.808	0.714	0.238	0.178	TSY4-2
BO 634	HS3 L WRIST VOLAR	950601	16.05	HS3 L W V	0.320	0.844	0.760	0.280	0.173	TSY4-3
BO 634	HS4 L WIRST, DORSUM	950601	16.06	HS4 L W D	0.170	0.647	0.545	0.545	0.213	TSY4-4
BO 634	NS1 L WRIST DORSUM	950601	16.07	HS4 L W D	0.250	0.920	0.895	0.316	0.145	TSY4-1NS
BO 634	NS1 L WRIST DORSUM	950601	16.07	NS1 L W D	0.250	0.920	0.895	0.316	0.145	TSY4-1NS
BO 634	NS2 L WRIST VOLAR	950601	16.09	NS2 L W V	0.490	1.000	0.978	0.089	0.041	TSY4-2NS
BO 634	HS1 R WRIST, VOLAR	950819	11.13	HS1 R WRIS	0.140	0.857	0.900	0.400	0.251	TSY6-1
BO 634	HS2 R WRIST, DORSUM	950819	11.14	HS2 R WRIS	0.400	0.900	0.909	0.212	0.130	TSY6-2
BO 634	HS3 L WRIST, VOLAR	950819	11.15	HS3 L WRIS	0.240	0.833	0.684	0.263	0.142	TSY6-3
BO 634	HS4 L WRIST, DORSUM, RADIA	950819	11.15	HS4 L WRIS	0.240	0.750	0.737	0.263	0.148	TSY6-4
BO 634	HS5 L WRIST,, DORSUM, ULNA	950819	11.16	HS5 L WRIS	0.220	0.818	0.647	0.294	0.176	TSY6-5
BO 634	NS L WRIST, VOLAR	950819	11.17	NS L WRIST	0.580	0.948	0.889	0.074	0.051	TSY6-5NS
BO 634	HS1 R WRIST, VENTRAL	951116	15.29	HS1 R WRIS	0.250	0.800	0.667	0.190	0.131	TSY7-1
BO 634	HS2 R WRIST, DORSUM	951116	15.30	HS2 R WR D	0.170	0.824	0.667	0.417	0.276	TSY7-2
BO 634	HS3 L WRIST VENTRAL	951116	15.31	HS3 L WR V	0.390	0.872	0.765	0.147	0.110	TSY7-3
BO 634	HS4 R WRIST DORSUM, RADIA	951116	15.32	HS4 L WR D	0.190	0.684	0.667	0.583	0.307	TSY7-4
BO 634	HS5 L WRIST, DORSUM, ULNA	951116	15.34	HS5 L WR V	0.180	0.833	0.750	0.500	0.261	TSY7-5
BS 570	NS1 GROIN	960330	11.53	NS1 GROIN	0.510	0.902	0.911	0.133	0.102	TSY14-N1
BS 570	NS2 L THIGH	960330	11.54	NS2 L THI	0.400	0.925	0.971	0.143	0.104	TSY14-N2
BS 570	HS2 R THIGH, MATURE SSG	960330	11.55	HS2 R THI	0.430	0.837	0.941	0.265	0.151	TSY14-2
BS 570	HS3 L THIGH,SSG	960330	11.56	HS3 L THI	0.260	0.769	0.650	0.300	0.169	TSY14-3
BS 570	HS4 L THIGH,DONOR,MATURE	960330	11.56	HS4 L THI	0.470	0.872	0.854	0.146	0.091	TSY14-4D
BS 570	HS, GROIN, inj	960330	11.57	HSinj	0.350	0.971	1.036	0.250	0.148	TSY14-ij
BO 634	NS1 L WRIST VENTRAL	960420	12.37	NS L VEN	0.430	0.907	0.868	0.132	0.089	TSY8-1
BO 634	NS2 R WRIST DORSUM	960420	12.38	NS2 R DOR	0.270	0.889	0.870	0.174	0.099	TSY8-N2
BO 634	HS1 R WRIST VOLAR, RADIAL	960420	12.39	HS1 R VOL	0.160	0.750	0.750	0.333	0.228	TSY8-1
BO 634	HS2 R WRIST VOLAR, ULNAR	960420	12.40	HS2 R V U	0.170	0.765	0.692	0.308	0.244	TSY8-2
BO 634	HS3 L WRIST DORSUM	960420	12.41	HS3 L DOR	0.180	0.889	0.714	0.286	0.145	TSY8-3
BO 634	HS4 L WRIST, VENTRAL, LAT	960420	12.42	HS4 L V L	0.220	0.773	0.500	0.222	0.132	TSY8-4
BO 634	HS5 L WRIST ULNAR	960420	12.43	HS5 L UL	0.080	0.750	1.000	0.600	0.604	TSY8-5

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 612	HS1 R ANKLE, ACTIVE, 1YR PO	1/5/95	13.24	HS1 R ANK	0.100	0.800	0.714	0.429	0.179	TWS1
PB 612	NS L ANKLE VS TWS1	1/5/95	13.26	NS L ANK	0.200	0.900	0.875	0.250	0.183	TWS2-NS
PB 612	HS3 R ANKLE, ACTIVE AND SU	1/5/95	13.27	HS3 R ANK	0.080	0.750	0.500	0.333	0.108	TWS3
PB 612	NS L ANKLE VS TWS3	1/5/95	13.29	NS L ANK	0.220	0.955	0.947	0.158	0.091	TWS4-NS
PB 612	R ANKLE-MEDIAL, RAISED,SOF	950302	16.00	R ANKLE-ME	0.060	0.667	0.667	1.000	0.414	TWS2-1
PB 612	R ANKLE-LATERAL, SOFT, FLA	950302	16.01	R ANKLE-LA	0.070	0.714	1.000	1.333	0.516	TWS2-2
PB 612	HS1 R FOOT, SOFT BUT RAISE	3/30/95	16.20	HS1 R FOOT	0.090	0.778	0.600	0.800	0.315	TWS4-1-2
PB 612	HS2 R FOOT, SOFT MILDLY RA	3/30/95	16.21	HS2 R FOOT	0.130	0.923	0.900	0.300	0.159	TWS4-2-1
PB 612	HS2 R FOOT, 2ND MX	3/30/95	16.22	HS2 R FOOT	0.140	0.786	0.875	0.750	0.316	TWS4-2-2
PB 612	NS L FOOT, VS HS2	3/30/95	16.26	NS L FOOT	0.470	0.915	1.000	0.146	0.120	TWS4-2NS
PB 612	NS L FOOT	5/15/95	0.49	NS L FOOT	0.220	0.955	0.947	0.158	0.091	TWS4-NS
PB 612	HS1, R FOOT,MEDIAL,ACTIVE,	950420	15.59	HS1 R FT M	0.140	0.929	0.750	0.167	0.074	TWS5-1
PB 612	HS2, R FOOT, LATERAL, SOFT	950420	16.00	HS2 R FT L	0.180	0.833	0.692	0.385	0.165	TWS5-2
PB 612	NS VS TWS5-1A	950420	16.02	L FT NS	0.570	1.000	1.163	0.163	0.104	TWS5-1N
PB 612	L FOOT, NS,V5 TWS5-2	950420	16.03	L FT NS	0.480	1.000	1.175	0.200	0.126	TWS5-2N
PB 612	HS1 R FOOT, MEDIAL	950518	16.03	HS1 R FOOT	0.130	0.923	0.818	0.182	0.081	TWS6-1
PB 612	HS2 R FOOT, LATERAL ASPEC	950518	16.05	HS1 R FOOT	0.230	0.913	0.889	0.278	0.151	TWS6-2
PB 612	NS1 L FOOT MEDIAL	950518	16.06	NS1 L FOOT	0.240	0.958	1.000	0.200	0.097	TWS6-1NS
PB 612	NS2 L FOOT LATERAL ASPECT	950518	16.07	NS1 L FOOT	0.330	0.909	1.000	0.179	0.087	TWS6-2NS
PB 612	HS1 R FOOT MEDIAL	950615	15.54	HS1 R FOOT	0.230	0.826	0.588	0.353	0.156	TWS7-1
PB 612	HS1 R FOOT MEDIAL	950615	15.55	HS1 R FOOT	0.090	0.778	0.500	0.500	0.175	TWS7-1B
PB 612	HS1 R FOOT MEDIAL	950615	15.56	HS1 R FOOT	0.080	0.750	0.600	0.600	0.337	TWS7-1C
PB 612	HS2 R FOOT LATERAL	950615	15.57	HS2 R FOOT	0.200	0.950	0.538	0.538	0.228	TWS7-2
PB 612	HS2 R FOOT, LATERAL	950615	15.58	HS2 R FOOT	0.230	0.913	0.867	0.533	0.233	TWS7-2B
PB 612	NS1 L FOOT MEDIAL	950615	15.59	NS L FOOT1	0.640	1.000	1.231	0.231	0.106	TWS7-1NS
PB 612	NS L FOOT LATERAL	950615	15.59	NS L FOOT2	0.560	0.964	0.981	0.077	0.052	TWS7-2NS
PB 612	HS1 R FOOT, MEDIAL	950713	15.36		0.160	0.563	0.556	0.778	0.366	TWS8-1
PB 612	HS2 R FOOT, LATERAL	950713	15.38	HS2 R FOOT	0.220	0.773	0.533	0.467	0.248	TWS8-2
PB 612	NS2 L FOOT, LATERAL	950713	15.40	NS2 L FOOT	0.630	0.984	0.932	0.068	0.060	TWS8-2NS
PB 612	NS1 L FOOT, MEDIAL	950713	15.41	NS1 L FOOT	0.740	0.986	1.015	0.121	0.075	TWS8-1NS
PB 612	HS1 R FOOT, MEDIAL	950713	15.42	HS1 R FOOT	0.110	0.727	0.571	0.571	0.293	TWS8-1B
PB 612	HS1 R ANKLE, MEDIAL	950907	16.31	HS1	0.090	0.778	0.833	0.500	0.326	TWS9-1
PB 612	NS1 L ANKLE FRONT	950907	16.32	NS1	0.850	1.000	0.875	0.063	0.039	TWS9-1NS
PB 612	HS2 R ANKLE, LATERAL	950907	16.32	HS2	0.460	1.000	1.150	0.150	0.098	TWS9-2
PB 612	NS2 L ANKLE VS HS2	950907	16.33	NS2	0.800	1.000	1.067	0.067	0.043	TWS9-2NS
PB612	HS1 R ANKLE, LATERAL	951005	16.18	HS1	0.160	0.750	0.500	0.333	0.153	TWS10-1
PB612	NS1 L ANKLE, LATEERAL	951005	16.19	NS1	0.710	1.000	0.954	0.092	0.063	TWS10-1N
PB612	HS2 R ANLKE, MEDIAL ASPEC	951005	16.19	HS2	0.230	1.000	1.353	0.353	0.171	TWS10-2
PB612	NS2 L ANKLE, MEDIAL	951005	16.20	NS1	0.820	1.000	1.108	0.108	0.064	TWS10-2N
PB612	HS1	951005	16.21	HS1	0.360	1.000	0.500	0.200	0.111	TWS10-1
PB612	HS2	951005	16.22	HS2	0.290	1.000	0.739	0.261	0.142	TWS10-2
PB 612	HS1 R FOOT, MEDIAL, STILL FI	951102	14.22	HS1 R FOOT	0.220	0.773	0.556	0.222	0.107	TWS10-1
PB 612	HS1 R FOOT,MED, STILL FIRM,	951102	14.22	HS1 R FOOT	0.220	0.773	0.556	0.222	0.107	TWS11-1
PB 612	HS2 R FOOT, LATERAL, MATU	951102	14.24	HS2 R FOOT	0.180	0.722	0.500	0.286	0.100	TWS11-2
PB 612	NS1 L FOOT, MED	951102	14.25	NS1 L FOOT	0.550	0.909	0.824	0.078	0.049	TWS11-1N
PB 612	NS2 L FOOT, LATERAL	951102	14.26	NS2 L FOOT	0.710	1.000	1.109	0.109	0.070	TWS11-2N
PB 612	HS1 R FOOT, LATERAL , MATU	951130	16.20	HS1 R FOOT	0.220	0.727	0.444	0.222	0.111	TWS12-1
PB 612	HS2 R FOOT, MEDIAL, STILL R	951130	16.21	HS1 R FOOT	0.180	0.778	0.643	0.286	0.164	TWS12-2
PB 612	NS1 L FOOT, LATERAL, VS HS1	951130	16.22	NS1 L FOOT	0.680	0.853	0.797	0.062	0.051	TWS12-N1
PB 612	NS2 L FOOT, MEDIAL VS HS2	951130	16.23	NS2 L FOOT	0.760	0.934	0.845	0.070	0.051	TWS12-N2
PB 612	HS1 R FOOT MEDIAL	960125	16.27	HS1 R FOOT	0.300	0.967	0.591	0.364	0.181	TWS13-1
PB 612	HS2 R FOOT LATERAL	960125	16.27	HS2 R FOOT	0.330	0.485	0.333	0.375	0.194	TWS13-2
PB 612	NS1 L FOOT, MEDIAL	960125	16.28	NS1 L FOOT	0.760	0.882	0.826	0.101	0.059	TWS13-N1
PB 612	NS2 L FOOT, LATERAL	960125	16.29	NS2 L FOOT	0.650	1.000	0.828	0.121	0.067	TWS13-N2
PB 612	HS1 R ANKLE LATERAL	960321	16.04	HS1 LAT	0.240	1.000	0.933	0.600	0.264	TWS14-1
PB 612	HS2	960321	16.05	HS2	0.210	0.571	0.467	0.400	0.188	TWS14-2
PB 612	NS1 LATERAL	960321	16.06	NS1	0.780	0.795	0.732	0.099	0.063	TWS14-N1
PB 612	NS2	960321	16.06	NS2M	0.760	1.000	1.134	0.134	0.074	TWS14-N2

[illegible]

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
BO 660	HS1 R THIGH, RIM OF SSG	1/5/95	10.23	HS1 R THI	0.140	0.857	0.900	0.400	0.236	WSS1
BO 660	HS2 L THIGH, MATURE DONAR	1/5/95	10.24	HS2 L THI	0.180	0.944	0.800	0.200	0.125	WSS2
BO 660	HS3 R KNEE, RIM OF SSG	1/5/95	10.26	HS3 R KNEE	0.100	0.800	0.625	0.250	0.119	WSS3
BO 660	NS R THIGH	1/5/95	10.27	NS R THI	0.280	0.929	1.000	0.217	0.151	WSS4-NS
BO 660	NS L THIGH, NEAR GROIN	1/5/95	10.28	NS L THI	0.420	0.952	1.000	0.167	0.112	WSS5-NS
BO 660	NS ON L KNEE VS WSS3	1/5/95	10.29	NS L KNEE	0.330	0.970	1.037	0.222	0.123	WSS6-NS
BO 660	HS1 R THIGH, SSG, 8/12 POST	3/11/95	10.42	HS1 R THI	0.220	0.818	0.778	0.222	0.157	WSS3-1
BO 660	HS2 R KNEE	3/11/95	10.42	HS2 R KNEE	0.140	0.786	0.800	0.400	0.173	WSS3-2
BO 660	HS3 L THIGH, DONAR	3/11/95	10.44	HS3 L THI	0.280	0.929	0.917	0.167	0.099	WSS3-3
BO 660	NS L THIGH, VS WSS3-1	3/11/95	10.46	NS L THI	0.380	0.921	1.000	0.187	0.122	WSS3-4NS
BO 660	NS L KNEE VS WSS3-2	3/11/95	10.47	NS L KNEE	0.270	0.926	0.913	0.174	0.100	WSS3-5NS
BO 660	NS L THIGH, VS WSS3-3, DON	3/11/95	10.48	NS L THI	0.380	0.974	1.097	0.226	0.155	WSS3-6
BO 660	NS L THIGH, VS DONAR WSS3-	3/11/95	10.48	NS L THI	0.380	0.974	1.097	0.226	0.155	WSS3-6NS
BO 660	HS1 R THIGH,MEDIAL,SSG,ACT	4/8/95	10.56	HS1 R THI	0.210	0.905	0.882	0.235	0.151	WSS4-1
BO 660	HS1 R THIGH, MEDIAL, SSG, 2	4/8/95	10.58	HS1 R THI	0.180	0.833	0.857	0.286	0.202	WSS4-1B
BO 660	HS1 R THIGH, MEDIAL,SSG,3R	4/8/95	10.58	HS1 R THI	0.190	0.895	0.867	0.267	0.168	WSS4-1C
BO 660	HS2 R KNEE, ACTIVE, RED	4/8/95	11.00	HS2 R KNEE	0.140	0.714	0.636	0.273	0.212	WSS4-2
BO 660	HS2 R KNEE MEDIAL, ACTIVE,	4/8/95	11.00	HS2 R KNEE	0.140	0.714	0.636	0.273	0.212	WSS4-2
BO 660	HS2 R KNEE,2ND MX	4/8/95	11.00	HS2 R KNEE	0.150	0.867	0.750	0.250	0.137	WSS4-2B
BO 660	HS2 R KNEE, 2ND MX	4/8/95	11.00	HS2 R KNEE	0.150	0.867	0.750	0.250	0.137	WSS4-2B
BO 660	HS2 R KNEE,3RD MX	4/8/95	11.01	HS2 R KNEE	0.140	0.929	0.818	0.273	0.179	WSS4-2C
BO 660	HS3 R CALF, RED	4/8/95	11.02	HS3 R CALF	0.200	0.850	0.688	0.250	0.152	WSS4-3
BO 660	HS3 R CALF, 3RD MX	4/8/95	11.03	HS3 R CALF	0.200	0.850	0.750	0.250	0.169	WSS4-3C
BO 660	HS3 R CALF,2ND MX	4/8/95	11.04	HS3 R CALF	0.170	0.765	0.643	0.214	0.136	WSS4-3B
BO 660	DONAR, MATURE, L THIGH	4/8/95	11.05	HSD L THI	0.290	0.931	0.960	0.160	0.143	WSS4-D
BO 660	DONAR, L THIGH,MATURE,2ND	4/8/95	11.06	HSD L THI	0.270	0.926	1.000	0.227	0.113	WSS4-DB
BO 660	DONAR, L THIGH, MATURE, 3R	4/8/95	11.07	HSD L THI	0.260	0.962	1.048	0.238	0.167	WSS4-DC
BO 660	NS L KNEE	4/8/95	11.09	NS L KNEE	0.220	0.909	0.944	0.222	0.108	WSS4-KNS
BO 660	NS L CALF	4/8/95	11.10	NS L CALF	0.230	0.913	0.947	0.211	0.137	WSS4-CNS
BO 660	R KNEE, HS2, ACTIVE, RED	950408	10.59	R KNEE,HS2	0.140	0.714	0.636	0.273	0.212	WSS4-2
BO 660	NS R THIGH,VS WSS4-1	5/15/95	0.34	NS R THI	0.280	0.929	1.000	0.217	0.151	WSS4-NS1
BO 660	HS1 SSG OF R THIGH, 8/12 PO	5/15/95	20.07	HS1 R THI	0.220	0.818	0.778	0.222	0.157	WSS3-1
BO 660	HS1 R THIGH, 11/12 POST-INJU	950610	10.34	HS1 R THI	0.250	0.920	0.952	0.190	0.113	WSS5-1
BO 660	HS2 R KNEE, STILL ACTIVE	950610	10.36	HS2 R KNEE	0.190	0.789	0.786	0.357	0.223	WSS5-2
BO 660	HS2 R KNEE, 2ND MX	950610	10.37	HS2 R KNEE	0.170	0.941	0.846	0.308	0.227	WSS5-2B
BO 660	HS3 L THIGH, DONOR SITE	950610	10.38	HS3 L THI	0.220	0.955	1.056	0.222	0.129	WSS5-3
BO 660	NS L THIGH,	950610	10.39	NS L THI	0.340	0.971	1.071	0.214	0.147	WSS5-1NS
BO 660	HS3 L THIGH, DONAR SITE	950610	10.39	HS3 L THI	0.310	0.903	0.960	0.240	0.140	WSS5-3B
BO 660	NS L KNEE	950610	10.40	NS L KNEE	0.350	0.914	1.000	0.167	0.102	WSS5-2NS
BO 660	HS1 R THIGH, SSG	950701	10.24	HS1 R THI	0.250	0.920	0.900	0.250	0.172	WSS6-1
BO 660	HS1 R THIGH, SSG, 2ND MX	950701	10.25	HS1 R THI	0.250	0.960	1.000	0.250	0.171	WSS6-1B
BO 660	HS2 R KNEE	950701	10.25	HS2 R KNEE	0.170	0.941	0.923	0.308	0.186	WSS6-2
BO 660	HS2 R KNEE, 2ND MX	950701	10.26	HS2 R KNEE	0.180	1.000	0.857	0.286	0.183	WSS6-2B
BO 660	HS3 L THIGH, DONOR SITE	950701	10.27	HS3 L THI	0.250	0.920	0.950	0.250	0.176	WSS6-3
BO 660	HS3 L THIGH, DONOR SITE, 2N	950701	10.28	HS3 L THI	0.250	0.920	1.050	0.250	0.177	WSS6-3B
BO 660	NS1 R THIGH,	950701	10.29	NS1 R THI	0.360	0.917	1.069	0.241	0.114	WSS6-1NS
BO 660	NS1 R THIGH	950701	10.30	NS1 R THI	0.340	0.941	1.034	0.172	0.097	WSS6-1N2
BO 660	NS2 L KNEE	950701	10.31	NS2 L KNEE	0.360	0.972	1.032	0.161	0.090	WSS6-2NS
BO 660	NS2 L KNEE, 2ND MX	950701	10.32	NS2 L KNEE	0.340	0.941	1.034	0.172	0.111	WSS6-2N2
BO 660	HS1 R THIGH, SSG, MATURE	950916	10.28	HS1 R THI	0.330	0.697	0.643	0.179	0.136	WSS7-1A
BO 660	HS1 R THIGH, MATURE SSG, 2	950916	10.29	HS1 R THI	0.400	0.925	0.714	0.143	0.092	WSS7-1B
BO 660	HS2 R KNEE, LATERAL	950916	10.30	HS2 R KNEE	0.260	1.000	1.000	0.182	0.135	WSS7-2A
BO 660	HS2 R KNEE, LATERAL	950916	10.31	HS2 R KNEE	0.250	0.920	0.800	0.250	0.169	WSS7-2B
BO 660	HS3 L THIGH, DONOR	950916	10.32	HS3 L THI	0.400	0.950	0.917	0.111	0.081	WSS7-3A
BO 660	HS3 L THIGH, DONOR	950916	10.32	HS3 L THI	0.520	0.865	0.771	0.083	0.041	WSS7-3B
BO 660	NS1 R THIGH	950916	10.33	NS1 R THI	0.450	0.933	0.900	0.125	0.082	WSS7-N1
BO 660	NS2 L KNEE, LATERAL	950916	10.34	NS2 L KNEE	0.420	0.857	0.737	0.105	0.070	WSS7-N2

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
PB 564	NS1 R HIP	950819	12.36	NS1 R HIP	0.370	0.919	1.000	0.194	0.140	YKL1-1NS
PB 564	HS1 L HIP, SURGICAL SCAR, 5/	950819	12.37	HS1 L HIP	0.160	0.688	0.800	0.600	0.358	YKL1-1
PB 564	HS2 L THIGH, DONOR, 5/52 PO	950819	12.39	HS2 L THI	0.360	0.917	0.935	0.161	0.105	YKL1-2
PB 564	HS3 L POPLITEAL, LATERAL, S	950819	12.39	HS3 L POPL	0.380	0.711	0.667	0.267	0.208	YKL1-3
PB 564	HS4 L KNEE LATERAL, SG	950819	12.41	HS4 L KNEE	0.490	1.000	1.485	0.485	0.206	YKL1-4
PB 564	HS6 R KNEE, MEDIAL	950819	12.43	HS6 R KNEE	0.190	1.000	0.533	0.267	0.149	YKL1-5
PB 564	NS2 R THIGH	950819	12.44	NS2 R THI	0.400	1.000	0.914	0.143	0.088	YKL1-2NS
PB 564	NS3 R POPLITEAL, LATERAL	950819	12.45	NS3 R POP	0.420	1.000	1.105	0.105	0.057	YKL1-3NS
PB 564	NS4 L KNEE, MEDIAL	950819	12.46	NS4 L KNEE	0.560	0.946	0.865	0.077	0.070	YKL1-4NS
PB 564	HS1 L HIP, DONOR	950930	11.41	HS1 L HIP	0.130	0.769	0.889	0.444	0.194	YKL2-1
PB 564	HS2 L THIGH, DONOR	950930	11.42	HS2 L THI	0.160	0.875	0.917	0.333	0.169	YKL2-1
PB 564	HS2 L THIGH, DONOR	950930	11.42	HS2 L THI	0.160	0.875	0.917	0.333	0.169	YKL2-2
PB 564	HS3 L POPLITEAL, SSG	950930	11.43	HS3 L POP	0.240	0.875	0.800	0.200	0.128	YKL2-3
PB 564	HS4 L LEG, SSG	950930	11.44	HS3 L LEG	0.280	0.893	0.818	0.273	0.178	YKL2-4
PB 564	HS5 R KNEE, CHRONIC SCAR,	950930	11.45	HS5 R KNEE	0.160	0.688	0.500	0.333	0.191	YKL2-5
PB 564	NS1 R HIP	950930	11.46	NS1 L HIP	0.360	0.972	1.030	0.091	0.070	YKL2-1NS
PB 564	NS2 R THIGH	950930	11.47	NS1 L HIP	0.490	0.939	0.956	0.089	0.079	YKL2-2NS
PB 564	NS3 R POPLITEAL	950930	11.48	NS3 R POP	0.480	1.000	1.143	0.143	0.090	YKL2-3NS
PB 564	NS4 R LEG	950930	11.48	NS4 R LEG	0.450	0.889	1.000	0.184	0.138	YKL2-4NS
PB 564	NS5 L LEG, MEDIAL	950930	11.49	NS5 L LEG	0.450	0.911	1.026	0.154	0.099	YKL2-5NS
PB 564	HS1 L HIP	951111	10.33	HS1 L HIP	0.170	1.000	0.818	0.545	0.281	YKL3-1
PB 564	HS2 L THI, DONOR	951111	10.34	HS2 L THI	0.490	0.776	0.750	0.114	0.082	YKL3-2
PB 564	HS3 L POPLITEAL	951111	10.35	HS3 L POP	0.160	0.750	0.667	0.333	0.260	YKL3-3
PB 564	HS4 L KNEE,	951111	10.36	HS4 L KNEE	0.190	0.684	0.571	0.357	0.245	YKL3-4
PB 564	HS5 R KNEE, MEDIAL	951111	10.37	HS5 R KNEE	0.300	0.600	0.417	0.250	0.153	YKL3-5
PB 564	HS1 R HIP, SURGICAL SCAR	951209	10.23	HS1 L HIP	0.350	1.000	0.889	0.296	0.134	YKL4-1
PB 564	HS2 L THIGH, DONOR, STOP P	951209	10.24	HS2 L THI	0.220	0.773	0.611	0.222	0.129	YKL4-2
PB 564	HS3 L POPLITEAL, SSG	951209	10.26	HS3 L POP	0.220	0.818	0.611	0.222	0.123	YKL4-3
PB 564	HS4 L KNEE SSG, SOFT	951209	10.26	HS4 L KNEE	0.480	0.813	0.738	0.143	0.094	YKL4-4
PB 564	HS5 R KNEE, CHRONIC SCAR	951209	10.27	HS5 R KNEE	0.300	1.000	0.480	0.200	0.097	YKL4-5
PB 564	HS1 L HIP	960106	10.52	HS1 L HIP	0.190	0.842	0.714	0.357	0.185	YKL5-1
PB 564	HS2 L THIGH, DONOR	960106	10.54	HS2 L THI	0.170	0.706	0.692	0.308	0.193	YKL5-2D
PB 564	HS3 L POPLITEAL	960106	10.55	HS3 L POP	0.190	0.842	0.857	0.357	0.238	YKL5-3
PB 564	HS4 L KNEE SSG	960106	10.56	HS4 L KNEE	0.210	0.762	0.706	0.235	0.200	YKL5-4
PB 564	HS5 R LEG, CHRONIC SCAR	960106	10.57	HS5 R LEG	0.170	1.000	1.417	0.417	0.174	YKL5-5
PB 564	NS1 R HIP	960106	10.58	NS1 R HIP	0.460	0.935	0.951	0.122	0.075	YKL5-N1
PB 564	NS2 R THIGH	960106	10.59	NS2 R THI	0.530	1.000	1.000	0.060	0.052	YKL5-N2
PB 564	NS3 R POPLITEAL	960106	11.00	NS3 R KNEE	0.430	1.000	1.103	0.103	0.103	YKL5-N3
PB 564	NS4 R KNEE	960106	11.01	NS4 R KNEE	0.420	1.000	1.079	0.105	0.083	YKL5-N4
PB 564	NS5 L LEG, MEDIAL	960106	11.02	NS5 L LEG	0.530	0.887	0.804	0.039	0.035	YKL5-N5
PB 564	HS1 L HIP, SURG SCAR	960203	10.13	HS1 L HIP	0.260	0.500	0.563	0.625	0.244	YKL6-1
PB 564	HS2 L THIGH, DONOR	960203	10.15	HS2 L THI	0.200	0.650	0.643	0.429	0.202	YKL6-2
PB 564	HS3 L POPLITEAL	960203	10.16	HS3 L POP	0.300	0.700	0.682	0.364	0.110	YKL6-3
PB 564	HS4 L KNEE, SUPPLE SG	960203	10.17	HS4 L KNEE	0.610	0.721	0.717	0.151	0.082	YKL5-4
PB 564	HS4 L KNEE, SUPPLE SG	960203	10.17	HS4 L KNEE	0.610	0.721	0.717	0.151	0.082	YKL6-4
PB 564	HS5 CHRONIC SCAR, R KNEE	960203	10.18	HS5 R KNEE	0.250	1.000	0.600	0.250	0.147	YKL6-5
PB 564	HS5 R LEG, CHRONIC SCAR	960203	10.18	HS5 R KNEE	0.250	1.000	0.600	0.250	0.147	YKL6-5
PB 564	NS1 R HIP	960203	10.20	NS1 R HIP	0.530	0.849	0.851	0.128	0.073	YKL6-N1
PB 564	NS2 R THIGH	960203	10.20	NS2 R THI	0.520	0.769	0.783	0.130	0.075	YKL6-N2
PB 564	NS3 R POPLITEAL	960203	10.21	NS2 R POP	0.560	1.000	1.040	0.120	0.085	YKL6-N3
PB 564	NS4 R KNEE	960203	10.22	NS4 R KNEE	0.580	1.000	1.094	0.094	0.058	YKL6-N4
PB 564	NS5 L KNEE MEDIAL	960203	10.23	NS5 L KNEE	0.480	0.958	1.024	0.143	0.098	YKL6-N5
PB 564	HS1 L HIP, SURGICAL SCAR	960316	9.46	HS1 L HIP	0.350	0.429	0.375	0.458	0.239	YKL7-1
PB 564	HS2 L THIGH, DONOR	960316	9.47	HS2 L THI	0.200	0.450	0.429	0.429	0.192	YKL7-2
PB 564	HS3 L POPLITEAL, SSG	960316	9.48	HS3 L POP	0.540	0.722	0.587	0.174	0.095	YKL7-3
PB 564	HS4 L KNEE, SSG	960316	9.49	HS4 L KNEE	0.920	1.000	0.938	0.136	0.094	YKL7-4
PB 564	HS5 R LEG, CHRONIC SCAR	960316	9.49	HS5 R LEG	0.310	0.484	0.320	0.240	0.110	YKL7-5
PB 564	NS1 R HIP	960316	9.50	NS1 R HIP	0.540	0.870	0.977	0.227	0.185	YKL7-N1
PB 564	NS2 L THIGH	960316	9.51	NS2 R THI	0.680	0.794	0.833	0.133	0.098	YKL7-N2
PB 564	NS3 R POPLITEAL	960316	9.52	NS3 R POP	0.580	1.000	0.980	0.160	0.111	YKL7-N3
PB 564	NS4 R KNEE	960316	9.53	NS4 R KNEE	0.610	0.754	0.732	0.089	0.068	YKL7-N4
PB 564	NS5 L LEG, MEDIAL	960316	9.53	NS5 L LEG	0.600	0.850	0.870	0.111	0.078	YKL7-N5
PB 586	NS1 R HIP	960518	10.32	NS1 R HIP	0.450	0.933	0.950	0.125	0.107	YKL8-N1
PB 586	HS1 L HIP	960518	10.33	HS1 L HIP	0.350	0.714	0.654	0.346	0.218	YKL8-1
PB 586	HS2 L THIGH, DONOR	960518	10.34	HS2 L THI	0.110	0.727	0.625	0.375	0.206	YKL8-2
PB 586	HS3 L POPLITEAL, RIM OF SSG	960518	10.35	HS3 L POP	0.220	0.818	0.778	0.222	0.147	YKL8-3
PB 586	HS4 L CALF, SSG	960518	10.36	HS4 L CALF	0.730	0.890	0.716	0.090	0.050	YKL8-4
PB 586	HS5 R CALF, CHRONIC	960518	10.36	HS5 R CALF	0.110	0.818	0.625	0.375	0.157	YKL8-5
PB 586	NS2 R THIGH	960518	10.37	NS2 R THI	0.370	0.946	0.970	0.121	0.099	YKL8-N2
PB 586	NS2 L CALF	960518	10.38	NS3 L CALF	0.380	0.974	1.000	0.118	0.084	YKL8-N3

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
BO 664	HS1 L LEG, ACTIVE 2/12 POST	1/3/95	9.41	HS1 L LEG	0.150	0.733	0.818	0.364	0.218	YYT1
BO 664	HS2 L LEG, ACTIVE SCAR, 2/12	1/3/95	9.43	HS2 L LEG	0.210	0.905	0.824	0.235	0.159	YYT2
BO 664	HS3, L ANKLE MEDIAL TO MAL	1/3/95	9.45	HS3 L MALL	0.130	0.769	0.667	0.444	0.320	YYT3
BO 664	HS4, R LEG, ACTIVE SCAR 2/12	1/3/95	9.48	HS4 R LEG	0.190	0.737	0.769	0.462	0.271	YYT4
BO 664	HS5 R LEG, ACTIVE SCAR	1/3/95	9.49	HS5 R LEG	0.120	0.917	0.778	0.333	0.178	YYT5
BO 664	NS AT R CALF	1/3/95	9.51	NS R LEG	0.400	0.950	1.000	0.143	0.084	YYT6-NS
PS 310	R ARM,SSG,2/12 POST-INJURY	5/11/95	23.50	HS1 R ARM	0.150	0.733	0.818	0.364	0.218	YYT1
BO 664	HS1 L LEG, ACTIVE, 2/12 POST	5/12/95	0.00	HS1 L LEG	0.150	0.733	0.818	0.364	0.218	YYT1
BO 650	HS1 L KNEE MEDIAL ASPECT	5/6/95	12.14	HS1 L KNEE	0.160	1.000	0.929	0.143	0.061	YYT1-S2
BO 650	HS2 L CALF MEDIAL ASPECT	5/6/95	12.17	HS2 L CALF	0.250	0.880	0.619	0.190	0.096	YYT2-S2
BO 650	HS3 ABOVE R TA	5/6/95	12.20	HS3 R TA	0.080	0.750	0.667	0.333	0.236	YYT3-S2
BO 650	HS4 ABOVE R LATERAL MALLE	5/6/95	12.22	HS4 R MAL	0.130	0.769	0.667	0.444	0.210	YYT4-S2
BO 650	HS1 L KNEE MEDIAL ASPECT	5/6/95	12.24	HS1 L KNEE	0.150	1.000	1.077	0.154	0.063	YYT1-S3
BO 650	HS2 L CALF MEDIAL ASPECT	5/6/95	12.27	HS2 L CALF	0.200	0.850	0.867	0.333	0.232	YYT2-S3
BO 650	HS3 ABOVE R TA	5/6/95	12.28	HS3 R TA	0.080	0.750	0.600	0.600	0.215	YYT3-S3
BO 650	HS4 ABOVE R LATERAL MALLE	5/6/95	12.32	HS4 R MAL	0.090	0.667	0.500	0.500	0.151	YYT4-S3
BO 664	L MEDIAL POPLITEAL, HS,	950216	17.14	L M-POPLIT	0.140	0.786	0.600	0.400	0.202	YYT2-1
BO 664	L CALF,HS	950216	17.17	L CALF,HS	0.140	0.857	0.818	0.273	0.171	YYT2-2
BO 664	BELOW L MEDIAL MALLEOLUS	950216	17.18	L MED MALL	0.160	0.813	0.583	0.333	0.234	YYT2-3
BO 664	HS, ON R FIBULA HEAD, ACTIV	950216	17.20	R FIBULA	0.150	0.800	0.667	0.250	0.115	YYT2-4
BO 664	HS ON R MED MALLEOLUS, ON	950216	17.22	R MED MALL	0.170	0.824	0.692	0.308	0.127	YYT2-5
BO 664	L MED POPLITEAL, HS1, ACTIV	950316	16.18	L LEG HS1	0.180	0.722	0.538	0.385	0.183	YYT3-1
BO 664	L CALF, HS2, MID-SHIN	950316	16.20	L LEG HS2	0.150	0.867	0.667	0.250	0.134	YYT3-2
BO 664	L ANKLE, BELOW MALLEOLUS	950316	16.22	L ANK HS3	0.180	0.778	0.538	0.385	0.221	YYT3-3
BO 664	R LEG, HS4 ON FIBULAR HEAD	950316	16.24	R LEG, FIB	0.130	0.769	0.600	0.300	0.211	YYT3-4
BO 664	R LEG, HS5, LAT TO MALLEOL	950316	16.26	R LEG, FIB	0.190	0.842	0.714	0.357	0.233	YYT3-5
BO 664	L MED POPLITEAL, HS1, ACTIV	950413	16.44	L POP,HS1	0.180	0.833	0.692	0.385	0.235	YYT4-1
BO 664	R MED POPLITEAL, NS	950413	16.46	R POP,NS	0.410	0.951	1.000	0.171	0.116	YYT4-1N
BO 664	L CALF, HS2, THIN	950413	16.47	L CALF,HS2	0.210	0.905	0.722	0.167	0.093	YYT4-2
BO 664	R CALF, NS	950413	16.48	R CALF,HS2	0.240	0.958	0.952	0.143	0.102	YYT4-2N
BO 664	L ANKLE, MEDIAL HS4	950413	16.50	L ANK,HS3	0.160	1.000	1.250	0.333	0.225	YYT4-2N
BO 664	L MEDIAL ANKLE, HS3	950413	16.50	L ANK,HS3	0.160	1.000	1.250	0.333	0.225	YYT4-3
BO 664	R MEDIAL ANKLE, NS	950413	16.51	R ANK,NS	0.250	1.000	1.250	0.250	0.142	YYT4-3
BO 664	R MEDIAL ANKLE, NS	950413	16.51	R ANK,NS	0.250	1.000	1.250	0.250	0.142	YYT4-3
BO 664	R MEDIAL ANKLE,NS	950413	16.51	R ANK,NS	0.250	1.000	1.250	0.250	0.142	YYT4-3N
BO 664	R FIBULAR HEAD, HS4	950413	16.52	R LEG, HS4	0.090	0.778	1.000	0.800	0.410	YYT4-4
BO 664	L FIBULAR HEAD, NS	950413	16.53	L LEG, NS	0.200	0.900	1.067	0.333	0.200	YYT4-4N
BO 664	R LATERAL TO LAT MALLEOLU	950413	16.55	R LAT,HS5	0.130	0.769	0.667	0.444	0.158	YYT4-5
BO 664	L LAT TO LAT MALLEOLUS, NS	950413	16.56	L LAT,NS	0.340	0.971	0.931	0.172	0.092	YYT4-5N
BO 664	HS1 L KNEE MEDIAL ASPECT	950506	11.22	HS1 L KNEE	0.150	1.000	1.167	0.250	0.207	YYT1-S1
BO 664	HS1 L KNEE MEDIAL ASPECT	950506	11.24	HS1 L KNEE	0.190	0.947	0.813	0.187	0.097	YYT1-S1
BO 664	HS2 L CALF MEDIAL	950506	11.25	HS2 L CALF	0.150	0.867	0.727	0.364	0.269	YYT2-S1
BO 664	HS3 R TA	950506	11.28	HS2 R TA	0.090	0.889	0.571	0.286	0.095	YYT3-S1
BO 664	HS4 R LAT MALLEOLUS	950506	11.31	HS4 R MAL	0.150	0.800	0.636	0.364	0.195	YYT4-S1
BO 664	HS1 L KNEE MED	950506	12.14	HS1 L KNEE	0.160	1.000	0.929	0.143	0.061	YYT1-S2
BO 664	HS2 L CALF MED	950506	12.17	L CALF MED	0.250	0.880	0.619	0.190	0.096	YYT2-S2
BO 664	HS3 R TA	950506	12.19	HS3 R TA	0.080	0.750	0.667	0.333	0.236	YYT3-S2
BO 664	HS4 ABOVE LAT MALLEOLUS	950506	12.21	HS4 R MAL	0.130	0.769	0.667	0.444	0.210	YYT4-S2
BO 664	HS1 L KNEE MEDIAL	950506	12.24	HS1 L KNEE	0.150	1.000	1.077	0.154	0.063	YYT1-S3
BO 664	HS2 L CALF MED	950506	12.26	HS2 L CALF	0.200	0.850	0.867	0.333	0.232	YYT2-S3
BO 664	HS3 R TA	950506	12.27	HS3 R TA	0.080	0.750	0.600	0.600	0.215	YYT3-S3
BO 664	HS4 ABOVE L LAT MALLEOLUS	950506	12.31	HS4 R MALL	0.090	0.667	0.500	0.500	0.151	YYT4-S3
BO 664	HS1 L KNEE, MEDIAL POPLITE	950518	18.10	HS1 L KNEE	0.120	0.750	0.667	0.333	0.230	YYT5-1
BO 664	HS2 L CALF	950518	18.11	HS2 L CALF	0.190	0.842	0.714	0.357	0.152	YYT5-2
BO 664	HS3 L ANKLE, BELOW MEDIAL	950518	18.12	HS2 L CALF	0.130	0.846	0.700	0.300	0.171	YYT5-3
BO 664	HS4 R FIBULAR HEAD	950518	18.13	HS4 R FIBU	0.140	0.786	0.700	0.400	0.246	YYT5-4
BO 664	HS5 LATERAL TO R LAT.MALLE	950518	18.14	HS4 R FIBU	0.070	0.714	0.500	0.167	0.164	YYT5-5
BO 664	NS R KNEE, MEDIAL POPLITEA	950518	18.15	NS1	0.290	0.931	1.000	0.208	0.135	YYT5-1NS
BO 664	NS2 R CALF,VS YYT5-2	950518	18.16	NS2 R CALF	0.240	0.958	1.000	0.200	0.122	YYT5-2NS
BO 664	NS3 R ANKLE, BELOW MEDIAL	950518	18.17	NS3 R ANK	0.080	0.875	0.571	0.143	0.063	YYT5-3NS
BO 664	NS4 L FIBULAR HEAD VS YYT5	950518	18.18	NS4 L FIBU	0.370	0.946	0.935	0.194	0.136	YYT5-4NS
BO 664	NS5 L ANKLE LAT TO LATERAL	950518	18.20	NS5 R ANK	0.290	0.966	0.917	0.208	0.104	YYT5-5NS
BO 664	HS1 L KNEE MEDIAL	950615	16.23	HS1 L KNEE	0.170	0.882	0.692	0.308	0.168	YYT6-1
BO 664	HS2 L LEG, MEDIAL	950615	16.24	HS2 L LEG	0.180	0.889	0.692	0.385	0.251	YYT6-2
BO 664	HS3 L TA	950615	16.24	HS2 L LEG	0.130	0.846	0.700	0.300	0.147	YYT6-3
BO 664	HS3 L TA	950615	16.24	HS3 L TA	0.130	0.846	0.700	0.300	0.147	YYT6-3
BO 664	HS4 R FIBULAR HEAD	950615	16.27	HS4 R FIB	0.080	0.875	0.667	0.333	0.319	YYT6-4
BO 664	HS5 R LATERAL MALLEOLUS	950615	16.27	HS5 R MALL	0.130	0.846	0.700	0.300	0.122	YYT6-5
BO 664	HS6 R ANKLE, BEHIND LAT MA	950615	16.29	HS6 R ANK	0.140	0.857	0.727	0.273	0.146	YYT6-6

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE
BO 664	HS1 L KNEE, MEDIAL POPLITE	950701	10.49	HS1 L KNEE	0.180	0.889	0.786	0.286	0.184	YYT7-1
BO 664	HS2 L CALF	950701	10.50	HS2 L CALF	0.260	0.885	0.739	0.130	0.077	YYT7-2
BO 664	HS3 L ANKLE, TA	950701	10.51	HS3 L TA	0.140	0.786	0.667	0.556	0.265	YYT7-3
BO 664	HS4 R FIBULAR HEAD	950701	10.52	HS4 R FIB	0.170	1.000	0.923	0.308	0.189	YYT7-4
BO 664	HS5 R ANKLE, LAT. TO LAT MA	950701	10.52	HS5 R ANK	0.180	1.000	0.786	0.286	0.130	YYT7-5
BO 664	NS R CALF	950701	10.54	NS R CALF	0.220	1.000	1.056	0.222	0.179	YYT7-NS
BO 664	HS1 L POPLITEAL	950727	16.36	HS1 L KNEE	0.170	0.765	0.615	0.308	0.235	YYT8-1
BO 664	HS2 L CALF	950727	16.37	HS2 L CALF	0.290	0.897	0.520	0.160	0.092	YYT8-2
BO 664	HS3 R FIBULAR HEAD	950727	16.38	HS3 R KNEE	0.140	0.857	0.727	0.273	0.145	YYT8-3
BO 664	HS4 R ANKLE, MEDIAL TO MED	950727	16.39	HS4 R ANK	0.150	1.000	1.250	0.250	0.157	YYT8-4
BO 664	HS1 L KNEE POPLITEAL	950921	16.36	HS1 L KNEE	0.210	0.714	0.471	0.235	0.156	YYT9-1
BO 664	HS2 L CALF	950921	16.37	HS2 L CALF	0.220	0.818	0.611	0.222	0.097	YYT9-2
BO 664	HS3 L ANKLE	950921	16.38	HS3 L ANK	0.240	0.875	0.474	0.263	0.170	YYT9-3
BO 664	HS4 R FIBULAR HEAD	950921	16.39	HS4 R FIB	0.190	0.789	0.625	0.187	0.104	YYT9-4
BO 664	HS5 LATERAL TO R LAT MALLE	950921	16.40	HS5 R LAT	0.190	1.000	1.188	0.187	0.092	YYT9-5
BO 664	HS1 L KNEE, POPLITEAL, SUP	951028	10.55	HS1 L KNEE	0.180	0.833	0.538	0.385	0.177	YYT10-1
BO 664	HS2 L CALF, NEAR MATURE	951028	10.57	HS2 L CALF	0.240	0.833	0.450	0.200	0.130	YYT10-2
BO 664	3 L BELOW MEDIAL MALLEOLU	951028	10.58	HS3 L ANK	0.290	0.552	0.273	0.318	0.167	YYT10-3
BO 664	HS4 L FIBULAR HEAD	951028	10.59	HS3 L ANK	0.290	1.000	0.565	0.261	0.136	YYT10-4
BO 664	HS5 LAT TO LAT MALLEOLUS	951028	11.00	HS5 R ANK	0.270	1.000	1.687	0.687	0.257	YYT10-5
BO 664	HS1 L KNEE, MEDIAL POPLITE	951116	15.50	HS1 L KNEE	0.200	0.800	0.625	0.250	0.100	YYT10-1
BO 664	HS1 L KNEE, MEDIAL POPLITE	951116	15.50	HS1 L KNEE	0.200	0.800	0.625	0.250	0.100	YYT11-1
BO 664	HS2 L CALF	951116	15.52	HS2 L CALF	0.360	1.000	1.059	0.059	0.030	YYT11-2
BO 664	NS, R CALF	951116	15.52	HS2 L CALF	0.510	0.784	0.739	0.109	0.088	YYT11-2N
BO 664	HS3 L ANKLE, BELOW MEDIAL	951116	15.53	HS3 L ANK	0.250	0.760	0.381	0.190	0.096	YYT11-3
BO 664	HS4 R FIBULAR HEAD	951116	15.54	HS4 R FIB	0.220	0.818	0.588	0.294	0.183	YYT11-4
BO 664	HS5 LATERAL TO R LATERAL	951116	15.55	HS5 R MAL	0.260	0.769	0.545	0.182	0.110	YYT11-5
BO 664	HS1 L POPLITEAL	951228	16.45	HS1 L KNEE	0.240	0.833	0.474	0.263	0.126	YYT12-1
BO 664	HS2 L CALF	951228	16.46	HS2 L CALF	0.270	0.852	0.583	0.125	0.097	YYT12-2
BO 664	HS3 L ANKLE, BELOW MEDIAL	951228	16.48	HS3 L ANK	0.280	1.000	1.217	0.217	0.117	YYT12-3
BO 664	HS4 R FIBULAR HEAD	951228	16.49	HS4 R FIB	0.250	0.760	0.476	0.190	0.100	YYT12-4
BO 664	HS5 R LAT TO LAT MALLEOLU	951228	16.50	HS5 R LAT	0.230	0.870	0.684	0.211	0.127	YYT12-5
BO 664	NS1 R KNEE MEDIAL POPLITE	951228	16.51	NS1 R KNEE	0.580	0.948	0.870	0.074	0.068	YYT12-1N
BO 664	NS2 R CALF	951228	16.52	NS2 R CALF	0.490	0.959	0.911	0.089	0.076	YYT12-2N
BO 664	NS3 R ANKLE BELOW MEDIAL	951228	16.52	NS3 R ANK	0.320	0.531	0.308	0.231	0.113	YYT12-3N
BO 664	NS4 L FIBULAR HEAD	951228	16.54	NS4 L FIB	0.120	0.833	0.778	0.333	0.178	YYT12-4N
BO 664	NS5 L ANKLE, LAT TO LAT MAL	951228	16.55	NS5 L ANK	0.390	0.923	0.727	0.182	0.136	YYT12-5N
BO 664	HS1 L MEDIAL POPLITEAL	960125	15.10	HS1 L POP	0.520	0.769	0.319	0.106	0.052	YYT13-1
BO 664	HS1 L MEDIAL POPLITEAL	960125	15.10	HS1 L POP	0.520	0.769	0.319	0.106	0.052	YYT13-1
BO 664	HS2 L CALF	960125	15.13	HS2 L CALF	0.390	0.846	0.618	0.147	0.093	YYT13-2
BO 664	HS3 BELOW L MEDIAL MALLE	960125	15.13	HS3 L ANK	0.200	0.700	0.400	0.333	0.140	YYT13-3
BO 664	HS4 R FIBULAR HEAD	960125	15.14	HS4 R FIB	0.230	0.739	0.500	0.150	0.095	YYT13-4
BO 664	HS5 LAT. TO R LAT MALLEOLU	960125	15.15	HS5 R ANK	0.210	1.000	1.750	0.750	0.322	YYT13-5
BO 664	NS1 R POPLITEAL	960125	15.16	NS1 R POP	0.660	1.000	0.934	0.082	0.054	YYT13-N1
BO 664	NS2 R CALF	960125	15.17	NS2 R CALF	0.450	0.867	0.750	0.125	0.090	YYT13-N2
BO 664	NS3 R ANK	960125	15.18	NS3 R ANK	0.320	1.000	0.654	0.231	0.149	YYT13-N3
BO 664	NS4 L FIBULAR HEAD	960125	15.19	NS3 R ANK	0.410	1.000	0.722	0.139	0.088	YYT13-N4
BO 664	NS5 L ANKLE,LAT	960125	15.20	NS5 L ANK	0.470	1.000	0.925	0.175	0.115	YYT13-N4
BO 664	NS5 L LAT MALL	960125	15.20	NS5 L ANK	0.470	1.000	0.925	0.175	0.115	YYT13-N5
BO 682	HS1 L MEDIAL POPLITEAL	960302	11.00	S1 L POPM	0.400	0.550	0.344	0.250	0.150	YYT14-1
BO 682	HS2 L CALF	960302	11.01	HS2 L CALF	0.450	0.689	0.564	0.154	0.082	YYT14-2
BO 682	HS3 L ANKLE	960302	11.02	HS3 L ANK	0.310	1.000	0.522	0.348	0.170	YYT14-3
BO 682	HS2 R FIBULAR HEAD	960302	11.03	HS4 R LEG	0.380	0.553	0.387	0.226	0.154	YYT14-4
BO 682	HS5 R ANKLE, LAT TO LAT MAL	960302	11.04	HS5 R ANK	0.320	1.000	1.391	0.391	0.183	YYT14-5
BO 664	NS1 R MEDIAL POPLITEAL	960302	11.05	NS1 R POP	0.730	0.849	0.818	0.106	0.068	YYT14-N1
BO 664	NS2 R CALF	960302	11.07	NS2 R CALF	0.460	1.000	1.103	0.179	0.110	YYT14-N2
BO 664	NS3 LAT TO L LAT MALLEOLUS	960302	11.08	NS3 L ANK	0.430	1.000	0.886	0.229	0.111	YYT14-N3
BO 664	NS4 L FIBULAR HEAD	960302	11.09	NS4 L FIB	0.560	1.000	0.878	0.143	0.097	YYT14-N4
BO 664	HS1 L POPLITEAL	960411	16.12	HS1 L POP	0.140	0.857	0.636	0.273	0.224	YYT15-1
BO 664	HS2 L ANKLE, MEDIAL	960411	16.13	HS2 L ANK	0.220	0.727	0.389	0.222	0.118	YYT15-2
BO 664	HS3 R ANKLE, LAT	960411	16.14	HS3 R ANK	0.230	0.913	0.684	0.211	0.159	YYT15-3
BO 664	HS4 R FIBULAR	960411	16.14	HS4 R FIB	0.170	0.824	0.692	0.308	0.173	YYT15-4
BO 664	NS1 R POPLITEAL	960411	16.15	NS1 R POP	0.470	0.979	0.977	0.068	0.051	YYT15-N1
BO 664	NS2 R ANKLE, MEDIAL	960411	16.16	NS2 R ANK	0.170	0.882	0.692	0.308	0.153	YYT15-N2
BO 664	NS3 L FIBULAR	960411	16.17	NS3 L FIB	0.330	0.939	0.900	0.100	0.063	YYT15-N3

NO	COMMENT	DATE	TIME	AREA	R0	R2	R5	R6	R8	PLOTFILE	
BO 664	HS1 L POPLITEAL	960509	17.38	HS1 L POP	0.180	0.833	0.500	0.286	0.169	YYT16-1	
BO 664	HS2 L ANKLE	960509	17.39	HS2 L ANK	0.240	0.875	0.550	0.200	0.122	YYT16-2	
BO 664	HS3 R MEDIAL MALLEOLUS	960509	17.40	HS3 R MALL	0.160	0.938	0.643	0.143	0.080	YYT16-3	
BO 664	HS4 R FIBULAR HEAD	960509	17.41	HS4 R FIB	0.170	0.765	0.538	0.308	0.364	YYT16-4	
BO 664	NS1 R POPLITEAL	960509	17.42	NS1 R POP	0.460	0.978	0.929	0.095	0.090	YYT16-N1	
BO 664	NS2 R ANKLE	960509	17.42	NS2 R ANK	0.280	0.929	0.708	0.167	0.090	YYT16-N2	
BO 664	NS3 L FIBULAR HEAD	960509	17.43	NS3 L FIB	0.190	0.947	0.800	0.267	0.156	YYT16-N3	
BO 664	HS1 L POPLITEAL	960606	16.17	HS1 L KNEE	0.230	0.739	0.421	0.211	0.102	YYT18-1	
BO 664	HS2 L CALF	960606	16.19	HS2 L CALF	0.230	0.870	0.700	0.150	0.073	YYT18-2	
BO 664	HS3 L ANKLE, MED MALL	960606	16.19	HS3 L ANK	0.160	0.813	0.636	0.455	0.205	YYT18-3	
BO 664	HS4 R FIBULAR HEAD	960606	16.20	HS4 L FIB	0.140	0.786	0.636	0.273	0.111	YYT18-4	
BO 664	HS5 R ANK	960606	16.21	HS5 R ANK	0.230	0.957	0.789	0.211	0.119	YYT18-5	
BO 664	NS1 R POPLITEAL	960606	16.22	NS1 R POP	0.560	0.946	0.865	0.077	0.077	YYT18-N1	
BO 664	NS2 R CALF	960606	16.23	NS2 R CALF	0.300	0.900	0.846	0.154	0.104	YYT18-N2	
BO 664	NS3 R AKNLE MEDIAL	960606	16.24	NS3 R ANK	0.270	0.926	0.696	0.174	0.109	YYT18-N3	
PB 588	HS1 R THIGH, PROXIMAL SSG	950916	10.51	HS1 R THI	0.290	1.000	0.833	0.208	0.155		
BO 682	HS1 R LEG	960302	10.36	HS1 R LEG	0.270	1.000	0.810	0.286	0.161		
PB 621	HS1 R ARM	960302	12.01	HS1 R ARM	0.470	0.872	0.707	0.146	0.070		
BO 684	HS2 R KNEE	960411	17.29	HS1 R KNEE	0.100	0.700	0.714	0.429	0.211		

O_PT	NAME	OT_CO	_SITE	SITE	T_1	T_2	T_3	T_4	T_5	T_6	T_7	T_8	T_9	T_10	T_11	T_12	T_13	T_14	T_15	T_16	T_17	T_18	_N1	_NLAST
1	SNF	PS318		1	14	2.0	1.0	1.0	2.0	2.0	2.0	2.0	3.0		3.0	2.0	2.0	2.0					.0	.0
1	SNF	PS318		2	14	.0	1.0	.0	.0	.0	.0	.0	.0		1.0	.0	1.0	.0					.0	.0
1	SNF	PS318		3	14	2.0	1.0	1.0	2.0	2.0	1.0	2.0	2.0		1.0	1.0	1.0	1.0					.0	.0
2	TWS	PB612		1	1	3.0	3.0	3.0	3.0	2.0	2.0	2.0	2.0	2.0	2.0	1.0		1.0				1.0	.0	.0
2	TWS	PB612		2	1	1.0	2.0	2.0	2.0	1.0	2.0	2.0	1.0	1.0	.0	1.0		.0				1.0	.0	.0
3	TSK	PB190		1	7	3.0		3.0			2.0	2.0	2.0	2.0	3.0		3.0				3.0		.0	.0
3	TSK	PB190		2	9	1.0		1.0			2.0	1.0	2.0	2.0	2.0		2.0				2.0		.0	.0
3	TSK	PB190		3	8	3.0		2.0		2.0	3.0	2.0	2.0		3.0		2.0				2.0		.0	.0
4	WCT	PS268		1	20	1.0				.0			1.0										.0	.0
4	WCT	PS268		2	17	3.0				1.0			2.0										.0	.0
4	WCT	PS268		3	16	1.0				1.0			1.0										.0	.0
4	WCT	PS268		4	7	2.5				1.0			1.5										.0	.0
5	CCC	PB621		1	7	2.0	2.0	1.0	1.0		2.0	1.0	1.0	.0		1.0	1.0	1.0			1.0	1.0	.0	.0
5	CCC	PB621		2	8	3.0	2.0	2.0	2.5	2.0	2.0	2.0	2.5	2.0		1.5	2.0	1.0			2.0	2.0	.0	.0
5	CCC	PB621		3	5	3.0	2.0	2.0	1.0		2.0	2.0	2.0	1.0		2.0	2.0	2.0			2.0	2.0	.0	.0
5	CCC	PB621		4	5	3.0	3.0	3.0	2.0		1.0	2.0	2.0	1.0		2.0	2.0	2.0			2.0	2.0	.0	.0
5	CCC	PB621		5	5	1.0	1.0	1.0	1.0		1.0	1.0	1.0			1.0	1.0				1.0	1.0	.0	.0
6	CSY	BS555		1	5	2.0	2.0			2.0	2.0		2.0	2.0	2.0	1.0	1.0						.0	.0
6	CSY	BS555		2	5	2.0	1.0			2.0	1.0		1.0		1.0	1.0	1.0						.0	.0
6	CSY	BS555		3	5	4.0	4.0			3.0	6.0	5.0	4.0	4.0	3.5	3.5	4.0						.0	.0
6	CSY	BS555		4	15	3.0	3.0			3.0	3.0	3.0	4.0	4.0	4.0	3.5	2.5	2.0					.0	.0
6	CSY	BS555		5	15	3.0	3.0			4.0	4.0	3.0	4.0	4.0	4.0	4.0	4.0						.0	.0
6	CSY	BS555		6	15	2.0	3.0			4.0	5.0	5.0		4.0	5.0	4.0	3.5	4.0					.0	.0
7	CTK	PB620		1	5	1.0				3.0	3.0		2.0	3.0	2.0	2.0	1.0	2.0	3.0	2.0	2.0	2.0	.0	.0
7	CTK	PB620		2	5	1.0						2.0	2.0	4.0	2.0	4.0	4.0	3.0	5.0	3.5	3.0	4.0	.0	.0
7	CTK	PB620		2	5	1.0			2.0	2.0		2.0	2.0	4.0	2.0	3.0	2.5		3.5	2.0	2.0	2.0	.0	.0
8	LSF	BO650		1	7	3.0	1.0	3.0		2.0								2.0		3.0	3.0	1.0	.0	.0
8	LSF	BO650		2	8	3.0	3.0	3.0		3.0			3.0	2.0	2.0	3.0		3.0		2.0	3.0	1.0	.0	.0
9	LSL	BO682		1	2	1.0	1.0	1.0	1.0	.0	.0		.0	.0	.0	.0		.0	.0	.0			.0	.0
9	LSL	BO682		2	3	1.0	1.0	1.0		1.0	1.0	.0	.0	.0	.0	.0	.0	.0	.0	.0			.0	.0
9	LSL	BO682		3	5	1.0	1.0	1.0	1.0	.0	1.0	1.0	1.0	.0	.0	1.0	.0	1.0	1.0	.0			.0	.0
9	LSL	BO682		4	5	1.0	1.0			.0	.0		.0	.0	.0	.0		.0	.0	.0			.0	.0
9	LSL	BO682		5	5	1.0	1.0	.0		.0	.0	.0	.0	.0	.0	.0		.0	.0	.0			.0	.0
9	LSL	BO682		6	3		1.0		1.0	1.0	1.0	1.0	.0	.0	.0	.0	1.0	.0	.0	.0			.0	.0
10	LYY	PB588		1	5	3.0				2.0	1.0	2.0	2.0	2.0	1.0	1.0	2.0	1.0		2.0	2.0	2.0	.0	.0
10	LYY	PB588		2	5	2.0					1.0	3.0	1.0	1.0		3.0	2.0	2.0	2.0	2.0	2.0	2.0	.0	.0
10	LYY	PB588		3	5	.0				.0	.0	.0	.0	.0	.0	1.0	.0	.0	.0	.0	.0	.0	.0	.0
10	LYY	PB588		4	5					3.0	2.0	2.0	2.0	1.0	.0	1.0	1.0	2.0	1.0	1.0	1.0	1.0	.0	.0
10	LYY	PB588		5	5						.0	.0		.0	.0	.0	.0	1.0	1.0	1.0	1.0	1.0	.0	.0

List of Graphs

graph	Title of graph	page
1	Thickness versus Clinical Grading	141
2	Mean R0 versus Clinical Grading	143
3	Mean R2 versus Clinical Grading	148
4	Mean R5 versus Clinical Grading	149
5	Mean R6 versus Clinical Grading	150
6	Mean R8 versus Clinical Grading	151
7	Application of V-T chart, case 11, YYT	174
8	Application of V-T chart, case 7, CTK	175
9	Application of V-T chart, case 10, LYY	176
10	Application of V-T chart, case 3, TSK	177
11	Application of V-T chart, case 14, TSC	178
12	Application of V-T chart, case 16, YKL (chronic scar on Rt leg)	179
13	Application of V-T chart, case 11, YYT (Lt lat. popliteal, SSG)	180
14	Application of V-T chart, case 11, YYT (Lt hip, donor site)	181

List of Figures

Figure	Description	page
1	Diagram of human skin	4
2	Epidermis from palm showing all of its layer	7
3	Schematic diagram showing the layer of the epidermis	9
4	Variation of elastic stiffness with age of skin	29
5	Langer's lines of the body	29
6	Stress/strain curves taken along and across Langer's lines	30
7	Load-extension curve of skin	32
8	Stress relaxation of skin	35
9	Stress-strain curve	36
10	Load-deformation test on human skin in vivo	41
11	Various human connective tissues in vitro in uniaxial tension	42
12	Human skin in vitro under repeated load cycles 1, 2 and 3	42
13	Uniaxial compression tests on human in vitro at varying strain rates	43
14	Force relaxation in human skin in vitro in uniaxial tension	43
15	Creep in human skin in vitro under uniaxial tension	44
16a	Cross-section to show general relation between surface appearance and ultimate depth of necrosis in typical cutaneous burns	51
16b	Surface appearance (degree) of burn correlates poorly with ultimate depth of necrosis	52
17	Depth of burns in relation to the layers of the skin	57
18	The Rule of Nine for estimation of surface area	61
19	Lund and Browder chart for accurate assessment of percentage body surface areas with age being considered	62
20	Sigmoid curves showing survival of humans as a function of total percentage of body surface burned and age	63
21	The three overlapping phases of wound repair	65
22	Chemical structure of pyridinoline	74

List of Figures

Figure	Description	
23	Compacted hyalinized collagen present in nodular formation	88
24	Nodular arrangement of hypertrophic scar	88
25	(a,b,c,d) Polarized light pictures of collagen fibers	99
26	Generation and reception of ultrasound	111
27	Ultrasonic equipment; circuit building blocks.	112
28	Ultrasound images (represent by electrical impulses) from normal skin and hypertrophic scar,	116
29	The suction device in the measurement of the elasticity	118
30	Strain-time curve of the cutometer	119
31	Diagram to show cross-section of normal skin	129
32	Diagram to show ultrasonograph of normal skin	129
33	Sketch of normal skin according to the ultrasonograph	129
34	Diagram to show ultrasonograph of scar tissue	129
35	Sketch of scar tissue according to the ultrasonograph	129
36	Strain-time curve with the deformation of the skin (strain) display as a function of time, and the R values calculated with the build-in database file (CT.dbf)	130
37	Illustration of the R8 value	132
38	Derivation of the equation of R8	132
39	Case 11, YYT	166
40	Case 7, CTK	167
41	Case 10, LYY	168
42	Case 3, TSK	169
43	Case 14, TSC	170
44	Case 16, YKL, chronic scar on Rt leg	171
45	Case 16, YKL, SSG on Lt lateral popliteal area	172
46	Case 16, YKL, donor skit on Lt hip	173

List of Tables

Table	Description of table	page
1	Comparison of the pathological classifications of burn injuries in terms of thickness	56
2	Coding of body parts	125
3	Assessment position	127
4	Clinical rating of burn scar (after Leung et al 1984)	133
5	Correlation of the measured thickness and elasticity with the clinical observation	136
6	Intra-examiner variability of ultrasonography and cutometer measurement	137
7	Average reading of normal skin from cutometer	138
8	Mean of thickness Vs clinical grading	140
9	Spearman correlation coefficients for each visco-elastic property	142
10	Mean of R0 Vs clinical grading	142
11	Mean of R2 Vs clinical grading	144
12	Mean of R5 Vs clinical grading	145
13	Mean of R6 Vs clinical grading	146
14	Mean of R8 Vs clinical grading	146
15	Summary of different measurements of hypertrophic scars	161
16	Elasticity of hypertrophic scar and the ultrasonographic presentation	162

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